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THE SURGICAL TREATMENT OF PULMONARY TUBERCULOSIS.[1]

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VOLTAIRE'S comment that the duty of a physician was to keep the patient amused while Nature cured him, was, until recently, true of the relation of physician to patient in pulmonary tuberculosis.(1) That it is no longer true is due to the introduction of collapse therapy and to the development of surgical methods for completing or for producing collapse of the lung.

In no disease is there more need for discrimination in selection of patients suitable for surgical treatment, and in no disease are the consequences of faulty judgement so distressing. Any discussion of the use of surgery in pulmonary tuberculosis necessarily entails a brief consideration of the pathology of the disease.

From the pathological point of view, patients with pulmonary tuberculosis fall into two groups: those who have had a primary infection in childhood, and those who have not. In the first group there appears to be some alteration in the reaction of the patient to subsequent infection with tuberculosis, so that the lung responds with a chronic inflammatory reaction in which fibrosis predominates as well as with an acute or subacute inflammatory reaction. The ratio of the fibrotic or chronic inflammatory response to the exudative or subacute inflammatory response varies with the general resistance of the patient. The disease runs a long course, advancing when resistance is low and retrogressing when it is high, until either the disease is overcome, or it destroys so much of the host that death occurs.

In the second group—those who have not been infected in childhood—the response to infection is an acute or subacute inflammatory one, with massive necrosis and caseation. There is no tendency to healing. The disease spreads rapidly and results in death in twelve to eighteen months. No means have yet been devised to influence favourably the course of the disease in this type of patient.

In dealing with resistant patients, however, much success has been won by the application of the lessons learned from a study of tuberculosis in general, and from an appreciation of the peculiar problems which exist in dealing with disease in the lung arising out of the mechanism of respiration.

The natural evolution of a tuberculous infection as seen under conditions easily observed and controlled, as when it affects bones and joints, has shown that it is a self-limiting process with a natural tendency to heal, unless fresh damage occurs. Three stages can be recognized: a stage of invasion, during which the healing processes of the body are less than the destructive processes of the disease; a stage of balance, in which the forces are roughly equal, though the healing processes gradually gain the upper hand; and a third stage in which the damage done is repaired. Each of these stages lasts about a year, but any reduction of general or local resistance will prolong them.

Overwork, exhaustion (physical or mental), starvation, exposure and intercurrent disease, in particular inflammatory and nutritional diseases, diminish the general resistance.

Local resistance is reduced by use of the affected part, which adds ordinary wear and tear to the ravages of disease, or by any other local disease.

In the lung, when general resistance is high, the virulence of the organisms low and the local resistance high, the infection may be immobilized by being walled off, or it may even be destroyed. If the local or general resistance

[1] Read at a meeting of the Queensland Branch of the British Medical Association on July 20, 1940, at Brisbane.

is low or the virulence high, advance of infection will occur.

The first result of the infection in the lung is the formation of a tuberculoma with a central area of necrosis or caseation surrounded by a zone of fibrosis. As this increases in size it erodes the bronchial tree and discharges the caseous material into it, leaving a cavity in the lung. This cavity has fibrous walls, and is lined by tuberculous granulation tissue. The exudate from the cavity is coughed up as sputum, and contains the mycobacterium. It may, however, be inhaled into other parts of the bronchial tree and set up a tuberculous bronchopneumonia, and thus spread the disease. The tuberculous area may go on spreading and erode vessels; this results in hæmoptysis or in the spread of the disease by blood vessels to other parts of the lung or the body. Secondary infection may reach the tuberculous area down the bronchi. As the resistance increases, there will be less tendency for concentric spread of the lesion, and spread along the bronchi, blood or lymph channels will have greater difficulty in establishing itself. Should either the local or general resistance be lowered, spread in all ways will be rapid.

The end result in the lung, therefore, is a combination of healing and spreading foci, complicated perhaps by the presence of a cavity.

This cavity, opening into a bronchus, is subject to the pressure in the bronchus—that is, almost atmospheric pressure. This is resisted by the intrapulmonary pressure on its walls. This pressure, exerted by the elastic tissue of the lungs, is equal to the difference between the pressure in the bronchi and the intrapleural pressure. Anything which brings the intrapleural pressure nearer the atmospheric pressure, will allow contraction of the elastic tissue of the lung. This contraction causes collapse of the lung and of the cavities.

In the absence of contraction of the lung the cavity remains open and may increase in size if there is loss of more lung tissue, or if there is a decrease in the intrapleural pressure. So long as the cavity is open, sputum persists, secondary infection may occur, and spread is certain sooner or later.

Closure of the cavity by natural means can only be the result of healing by granulation—a long process, impossible of achievement in the case of large cavities. If the walls can be brought together, or into contact, the natural process of healing is at once facilitated. Moreover, the reduction in sputum and the diminution in the area open to secondary infection improve the general condition, and consequently the resistance, and a benign circle is started.

The ideal method of treatment in any form of tuberculosis is complete eradication of the disease, and there is no way of attaining this as yet, other than by surgical means. This is possible only when the disease is confined to an organ or part of an organ, loss of which can be borne by the body, and when the patient is able to withstand the strain of the necessary operation.

As these conditions are but rarely fulfilled, it follows from the foregoing consideration of the disease process, that the aims of treatment are to aid the natural processes of healing. There are, then, three principles in treatment: (i) the general resistance must be maintained or, if possible, increased; (ii) the local resistance must be maintained or, if possible, increased; (iii) the local processes of dealing with the disease must be assisted in every way possible.

Improvement in the general resistance is secured by the removal of any of the known causes of loss of resistance.

The patient must be prevented from overwork, and by overwork is meant any activity which causes fatigue. The amount of work which may be done is judged entirely by this criterion. Exhaustion of any type must be eliminated. Starvation, exposure and worry are the usual causes, and of these neither starvation nor exposure should exist. Worry is usually due to economic factors which cannot be eliminated without some scheme of assistance supported by the State.

Any intercurrent disease must be eradicated if possible, or if this is not possible, as in *diabetes mellitus*, should be balanced. Any source of infection should be removed; the teeth, often septic, should receive adequate attention. Mullin[2] has shown experimentally that infection of the paranasal sinuses is often followed by enlargement of the lymph glands at the bifurcation of the trachea, and this may cause secondary effects in the bronchi due to obstruction. It follows that every focus of infection in these sinuses must be removed.

Local rest is the chief means by which local resistance can be improved. In the case of the lungs, this is not easily obtained. Any use of the body as a whole involves use of the lungs. This use must therefore be reduced to a minimum so long as any active disease is present. This involves rest, and to be of value it must be the maximum—that is, rest in bed, the so-called absolute rest—so long as any pyrexia, tachycardia or lassitude is present.

Any direct means of resting the affected parts of the lungs must involve the prevention of their use. This can be effected by blocking of the bronchus to the area, or by the prevention of that difference between intrapleural and atmospheric pressure which enables respiration to occur. The first method is rarely applicable, though in the case of localized cavitation, blocking of the bronchus by a balloon as devised by Brooks[3] has been attempted.

There remain means of preventing the development of a negative intrapleural pressure. This can be done by the injection of a gas into the cavity inside the rigid chest wall—that is, into the pleural cavity or into the extrapleural space, by destruction of the rigidity of the chest wall by the removal of ribs, or by the production of paralysis of the diaphragm.

All these methods result in the reduction of the negative pressure in the intrapleural cavity, and so in collapse of the lung. In the case of the first two procedures, intrapleural and extrapleural pneumothorax, the contraction of the lung will exist only so long as the negative pressure in the pleural cavity is reduced, so that the air must be renewed as fast as it is absorbed.

After the third procedure, removal of the ribs, the loss of rigidity of the chest allows the lung to contract. If the ribs are removed subperiosteally, the rib beds collapse onto the subjacent contracted lung. Regeneration of the ribs in this position results in a rigid chest wall in the new position. This means that the maximum expansion of the lung is fixed at the contracted position taken up by it after removal of the ribs. It is therefore a permanent and irreversible change.

Paralysis of the diaphragm, while it reduces the intrapleural negative pressure, does so only to a very limited degree. It diminishes the ability to cough, as does thoracoplasty until the ribs regenerate.

For the success of any form of collapse therapy the whole of the diseased area must share in the contraction of the lung, and this collapse must be maintained until healing is complete.

Success in the use of artificial pneumothorax depends on the securing of complete collapse and in its maintenance until healing is complete. The latter is a matter of supervision only, but complete collapse may be prevented by the presence of adhesions between the lung and the chest wall.

There are four types of adhesions: (i) massive adhesions of such an extent that the pleural cavity is almost or completely obliterated, and no collapse can be obtained; (ii) close union of the visceral and parietal pleura in broad and short bands; (iii) fine string-like adhesions, and long bands formed by the parietal and visceral pleura only; (iv) fine string-like adhesions and long bands formed chiefly of pleura, but containing in their base a core of lung tissue. By the use of a thoracoscope and cautery all of these except the first can be divided.

The thoracoscope is inserted into the chest through a cannula thrust between the ribs under local anæsthesia. In some instruments an operating attachment enables a cautery to be used through the thoracoscope. The alternative method involves the use of two cannulæ, the thoraco-

scope passing through one and the cautery through the other. This has the decided advantage that the adhesions can be inspected from two sides by using the thoracoscope through both cannulæ, and that the second cannula can be placed so that access to the adhesions is easy.

For successful division, adhesions must be accessible. They must be long enough to allow division without risk of coagulation spreading to neighbouring vessels. They must be divisible without risk of injury to the lung. Only the string-like adhesions and long bands satisfy these requirements. When there is any possibility that they contain a core of lung, they should be severed or enucleated by division of their attachment to the chest wall. The core occasionally is rendered visible by trans-illumination of the adhesion. When the adhesions are short or are attached to pleura overlying the subclavian artery or the mediastinal vessels, division entails great risk.

Although inspection through a thoracoscope is the only certain means of deciding whether or not the adhesions are suitable for division, X-ray examination will show in which cases it is worth while inserting a thoracoscope.

The procedure is often lengthy and requires great patience in the surgeon and the patient. Apart from this, the only other requirement is skill in placing the cannulæ so that there is room to manœuvre the cautery into contact with the adhesions.

There are many complications, but apart from the effusion of clear fluid, they are infrequent.

There may be bleeding from intercostal vessels damaged during insertion of the cannulæ. This may be of such volume that the bleeding point must be exposed and secured. Division of adhesions, therefore, should be attempted only by a surgeon capable of a speedy thoracotomy and in a place suitably prepared for it. There may be bleeding also from vessels in the divided adhesions. This is usually slow, and results in the gradual accumulation of blood in the pleural cavity. It may continue for so long and to such an extent that blood transfusion is indicated to replace the loss. In about one-third of all cases a pleural effusion of clear sterile fluid occurs.[4] The treatment of effusion, whether of fluid or of blood, is repeated aspiration until the pleural cavity is emptied and remains empty. The fluid may not collect for some weeks, but more commonly does so in the first fortnight.

Infection of the effusion either by *Mycobacterium tuberculosis* or by pyogenic organisms may occur. Both are serious complications. Streptococcal infections may sometimes be controlled by the use of drugs of the sulphanilamide group, but at best fresh adhesions usually result. At worst, there is an empyema. Infection with tuberculosis also means an empyema. Treatment of both types of empyema conflicts with treatment of the pulmonary condition, for to deal with the empyema the pleural cavity must be obliterated; but treatment of the empyema must prevail. This is drainage either by aspiration or intercostal tube with constant negative pressure, followed by obliteration of the cavity by thoracoplasty, which will enable the collapse of the lung to be maintained.

The cause of the effusions is not fully understood. Prolonged and extensive operations and injury to the lung all seem to play a part in the cause. Infections may arise from the same factors. Thus repeated short sessions are wiser than one long session at which many adhesions are divided.

The rate of air absorption from the pleural cavity may be increased for a few days after the operation. Air may escape from the pleural cavity into the subcutaneous tissues, where it produces surgical emphysema. The end result in either case is that the lung may reexpand. If it comes into contact with the chest wall and remains there for more than a very few hours, new adhesions will form with loss of the pleural space and the pneumothorax. Refills must be given just as if the pneumothorax had been freshly induced on the day of operation.

Separation of the parietal pleura from the chest wall will also allow the lung to contract, so long as the parietal pleura is kept from returning to its original site. The first method devised to keep the pleura in its separated position was the introduction of air and fluid. Then wax was tried, and now air is again being used. All methods involve pressure on the parietal portion of the pleura, and, through it, on the lung, for unless the air is kept at a positive pressure, the collapse cannot be maintained. This pressure may cause necrosis of the tissues over the cavity in the lung and communication between the cavity and the extrapleural space with infection by either tuberculosis or pyogenic organisms, or both. Wax, in addition to causing pressure necrosis, may be extruded into the cavity and be coughed up, or it may move. In either case the lung may reexpand. As in many cases one of these complications ensues, the use of wax has been abandoned. At the present time air under positive pressure or oil is used.

The separation can be done only by direct means, and is usually accomplished through a posterior incision over the third or fourth rib. A piece of one of these ribs about three inches long is resected subperiosteally, back to the transverse process. The extrapleural space is defined by cautious dissection through the periosteum. The pleura is then separated from the chest wall under direct vision by a combination of blunt and sharp dissection. An illuminated dissector is a great aid in this step.

The separation of the pleura is carried out to such an extent as will allow all of the diseased lung to contract. Separation is possible as low as the hilum medially and down to the diaphragm laterally. The separation carried out is slightly greater than is required by the extent of the disease, as there is always some reattachment of the parietal portion of the pleura to its bed. Numerous small vessels are encountered and all bleeding must be controlled. By the use of diathermy or silver clips any bleeding point can be secured. The incision is closed and made as airtight as possible by careful stitching and by approximation of the neighbouring ribs by pericostal sutures.

Air is introduced into the extrapleural space at once until a positive pressure of about 10 to 15 centimetres of water is produced. Refills are given the same evening and each day for two days. The refills are then spread out, the length of the intervals being gauged by the X-ray appearances.

It is usual for air to be forced into the tissues for the first few days. This causes a good deal of soreness wherever the air is, but seldom has any serious effects.

During operation two accidents may happen. The pleura may be torn or the cavity in the lung may be opened. The first is not very serious if the patient's vital capacity is sufficient to support the open pneumothorax. Sometimes it can be closed, and, if so, the operation should be continued. If the opening cannot be closed, it is probably wiser to abandon the operation. If, however, the cavity in the lung is opened, as may result during attempts to separate firmly adherent pleura, the operation area must be infected from it. The operation should be abandoned at once. If the patient survives, a thoracoplasty can be attempted later. If very firm adhesions are found between the chest wall and the parietal pleura, the operation should be terminated, as the risk of damaging the lung is great. Such adhesions occur when there is much fibrosis of the apex of the lung with displacement of the trachea to the affected side.

Some shock may follow the operation. This is seldom severe, and recovery is quick.

There is always some bleeding into the extrapleural space, which may be of such an amount and so rapid that transfusion of blood is required urgently immediately after the operation. However, the usual thing is for the blood to accumulate slowly. It then occupies the lowest part of the space and tends to keep the pleura down. If it is not removed, organization will occur, and the pleura will become reattached to the chest wall. Removal of this fluid presents several problems. There are many small pieces of fibrin in it which block a small aspirating needle. If a large needle is used, air under positive pressure may escape, or organisms may reach the space

along the needle track. The upper surface of the parietal portion of the pleura immediately after operation is concave, forming a central lake. Surrounding this is the pleura attached at a higher level than the centre. Under the pleura is a layer of lung. The blood fills the lake. If aspiration is attempted from a level on the chest wall corresponding with the bottom of the lake, the needle must pass through the lung. On the withdrawal of the needle, air may rush into the lung and produce an air embolus. Thus aspiration must be from above with a long needle. As the patient usually is somewhat sore from surgical emphysema, the procedure is by no means easy. The bleeding may persist, rendering frequent aspirations necessary, and if a considerable volume of blood is lost over several days, transfusion will be required.

Effusions of clear fluid may occur soon after operation or sometimes after several weeks. While the fluid may be absorbed, it is better to remove it by aspiration.

Infection of the extrapleural space is unfortunately not uncommon. Undoubtedly in many cases this is due to necrosis of the tissues over the cavity with formation of a fistula into a bronchus. But it may arise from damage to the lymph glands found between the pleura and the chest wall, which, as Van Hazel has shown, often contain areas of tuberculous or chronic non-specific inflammatory tissue.[5] Infection may also result if the cavity has been opened at operation. Whatever the cause, the space must be drained, and as in infection of a pneumothorax, the only treatment offering any possibility of cure is thoracoplasty.

In some cases in which artificial pneumothorax has been carried out, collapse is prevented by a mass of apical adhesions—adhesions too short or too extensive to be divided by thoracoscope and cautery, and yet well clear of the mediastinum. Formerly the only way to deal with these cases was to abandon the pneumothorax and substitute a thoracoplasty. Extrapleural separation from the chest wall of the parietal pleura to which the lung is adherent, will leave as the only attachments of the lung to the chest wall a ring of parietal pleura; this ring separates the extrapleural space from the intrapleural pneumothorax. A circular cut through this parietal pleura, completely severing the area of adhesions from that attached to the chest wall, will allow the lung with the adhesions and two layers of pleura to fall away from the chest wall. It is, in fact, an extrapleural division of adhesions. This operation is carried out in the same way as an extrapleural pneumothorax until the area of adhesions is completely clear of the chest wall. A circular cut can be made at once or alternatively the extrapleural pneumothorax is carried on for ten to fourteen days, and the division made with a thoracoscope and cautery inserted into the pleural cavity.

With the first procedure, emphysema of the chest wall usually results, but as a negative pressure will maintain the collapse of the lung, this is less than with an extrapleural pneumothorax. There is always an effusion of fluid, consisting chiefly of blood. As this collects at the bottom of the pleural cavity, it is easily aspirated as access is good and well away from the operation area. Several aspirations may be necessary before the pleural cavity will become dry and remain so.

The second procedure is subject to all the complications of an extrapleural pneumothorax during the first ten to fourteen days. The second step may also result in an effusion into the pleural cavity, so that the advantages of this procedure are in my opinion very doubtful.

Destruction of the rigidity of the chest wall by the removal of ribs will prevent the development of a negative pressure within the chest, but so long as rigidity is absent, respiration will cause paradoxical movement of this part of the chest wall and the underlying lung. This will not result in rest to the lung, although contraction can take place. The paradoxical movement of the lung may lead to aspiration of infected sputum into the other lung. The rigidity of the chest wall must be restored, and if the ribs are removed by subperiosteal resection, their regeneration in the new collapsed position will restore it. It follows then that an operation on the ribs or thoracoplasty entails subperiosteal removal of the ribs and maintenance of the rigidity of the chest wall by external pressure, which also keeps the rib beds in contact with the contracted lung, until regeneration occurs in two or three weeks.

Thoracoplasty has undergone a steady revolution since it was first performed in 1888 by Bier and von Mickulicz, who resected ribs over the site of the cavity.[6] This gave unsatisfactory results, and the next step was the removal of enough ribs to allow contraction of all the diseased lung tissue. The extent of rib resection has gradually increased as experience showed that more and more contraction of the lung was required in order that collapse of the cavities should follow.

It was found that the two areas in which cavities persisted owing to inability of the lung containing them to contract, were the posterior portion of the lung which lies in the gutter lateral to the vertebral column, and the apex of the lung. The first was remedied by resection of the ribs medial to the transverse processes; the depth of this gutter was thus altered. Removal of the transverse processes themselves, while it does not increase the amount of contraction possible, does greatly increase the post-operative deformity, and is not therefore worth while. Failure of the apex to contract has been overcome by the operation devised by Semb,[7] who in addition to carrying out an ordinary thoracoplasty, divides all the structures which pass from the neck and vertebral column to the upper three rib beds and chest wall over the dome of the pleura. These attachments are the muscles inserted into the ribs, the anterior and medial scalenes, the periosteum and intercostal muscles, and various strips of fascia, the most pronounced of which are those attaching the dome of the pleura to the brachial plexus, particularly posteriorly, and those between the dome and the subclavian vessels. These include the fasciæ described by Sibson and Sibelcau. This procedure of extrafascial apicolysis has greatly increased the possible contraction of the apex.

Evolution has also occurred in the direction of lessening the severity of the operation and the mechanical disorder occasioned by it, by division of the operation into stages, and by the provision of external pressure by a pad to act as a rigid chest wall until there is regeneration of the ribs. The stages are described as upper, middle and lower, and each of these may be further divided. The number of ribs removed at each operation varies from one or two to five or six.

The ideal anæsthetic for extrapleural pneumothorax or for thoracoplasty is one that will preserve the cough reflex, reduce the respiratory movements to a minimum and provide anæsthesia, without causing any damage to the diseased area. While local anæsthesia fills most of these requirements, there is always some discomfort associated with its use,[8] and when several operations have to be performed, patients are not always willing to have local anæsthesia. There remains cyclopropane or nitrous oxide, combined with basal anæsthesia. Cyclopropane is almost ideal, for although it abolishes the cough reflex, the respiratory movements are reduced to a minimum.

For the upper stage a vertical paravertebral incision is made, curving round the inferior angle of the scapula. It begins opposite the spine of the second dorsal vertebra. The muscles attaching the scapula to the chest wall are divided, the scapula is retracted, and the attachments of the *serratus anterior* to the upper ribs are divided. This exposes the whole length of the first and second ribs and the greater part of the third rib. The third rib is removed subperiosteally from the nipple line anteriorly to the point just in front of the transverse process posteriorly after division of the ligaments of the costo-transverse joint. The second rib is removed within the same limits. The first rib is cleared and divided subperiosteally at the transverse process; the first dorsal nerve must be avoided while this is being done. The free end of the anterior portion of the rib is pulled down; the rib can thus be cleared to the costal cartilage, at which place it is divided and removed.

If an extrafascial apicolysis is to be added, the scalenes are divided at their attachments to the ribs. The periosteum

ILLUSTRATIONS TO THE ARTICLE BY DR. A. J. CANNY.

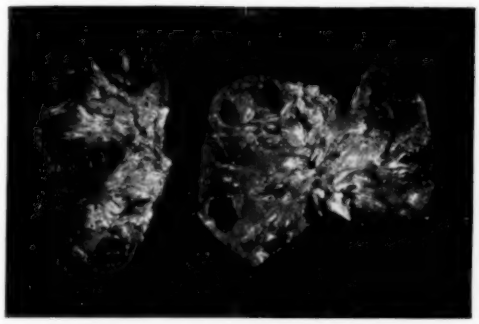

FIGURE I: Case I. Senile nephrosclerosis. (Reduced to half normal diameter.) In the greatly contracted kidneys the prominent thickened walls of the branches of the renal artery can be distinguished. Both cortex and medulla are much reduced in bulk, and several cysts are present in the parenchyma.

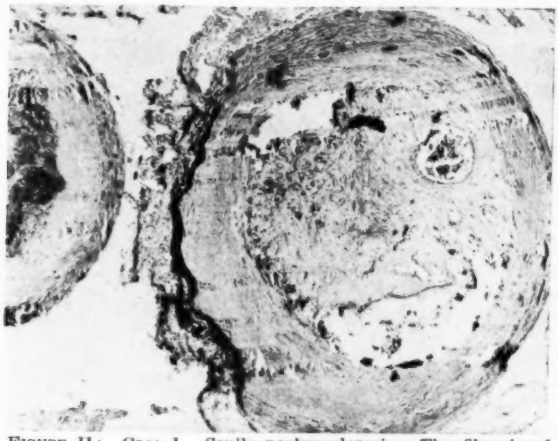

FIGURE II: Case I. Senile nephrosclerosis. The fibrosis of the medial coat of the primary branches of the renal artery is shown in the right-hand vessel. Occlusion of the lumen of this artery has been produced by thrombosis associated with an area of atheroma, but during organization of the thrombus recanalization has occurred. (×34.)

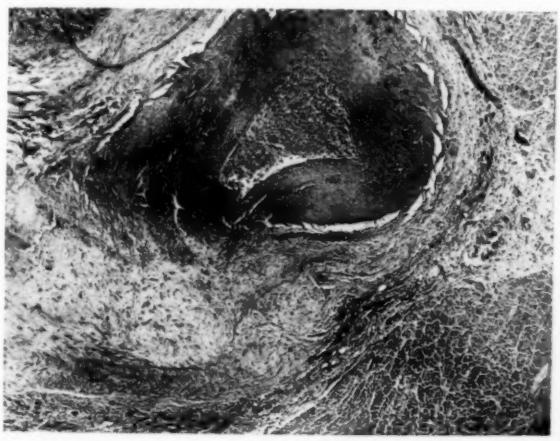

FIGURE III: Case II. *Polyarteritis nodosa.* An aneurysm of the coronary artery is partly filled with blood clot. The muscular wall of the artery can be distinguished towards the lower left-hand portion of the photograph. Towards the right, two areas of myocardium can be distinguished. (× 43.)

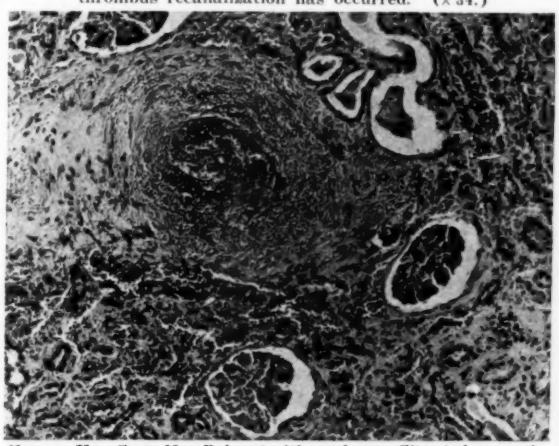

FIGURE V: Case II. *Polyarteritis nodosa.* Situated near the centre is a necrotic interlobular arteriole, the walls of which are infiltrated with blood. Towards the right the blood has spread slightly into the interstitial tissue. (×124.)

FIGURE IV: Case II. *Polyarteritis nodosa.* (Reduced to half normal diameter.) The subcapsular surface of one kidney is coarsely mottled and flecked with hæmorrhagic spots. The cut surface of the other kidney shows a number of small hæmorrhages, and the large subcapsular effusion of blood has caused some deformation. The channel along which the blood apparently extended from within the renal tissue is seen towards the upper pole.

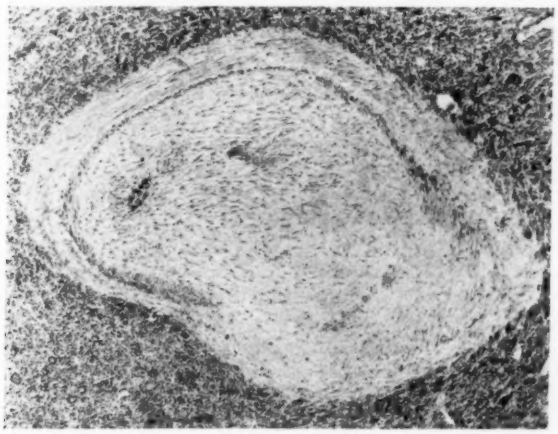

FIGURE VI: Case II. *Polyarteritis nodosa.* A small branch of the renal artery occluded by a great proliferation of fibrocellular connective tissue. The muscular wall has been largely replaced by fibrous tissue and in one area has disappeared. At this point the distortion of the wall suggests abortive aneurysm formation. (×96.)

ILLUSTRATIONS TO THE ARTICLE BY DR. A. J. CANNY.

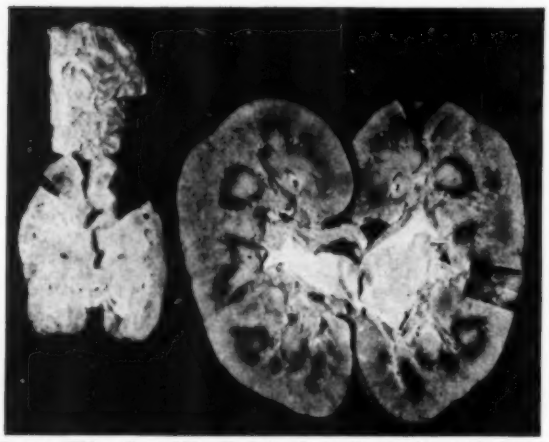

FIGURE VII: Case III. Unilateral renal atrophy. (Reduced to three-eighths normal diameter.) The atrophic left kidney is surmounted by a suprarenal gland of normal size. On the right is seen the enlarged kidney, the vessels of which showed changes suggestive of malignant nephrosclerosis.

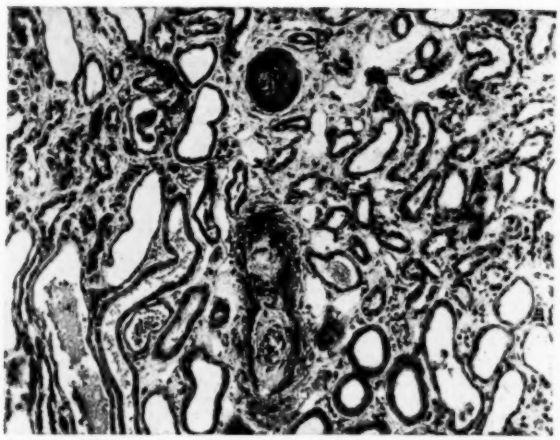

FIGURE VIII: Case III. Compensatory hypertrophy with malignant nephrosclerosis. The illustration shows early necrosis of the wall of an interlobular arteriole. (×124.)

FIGURE IX: Case IV. Chronic pyelonephritis. (Reduced to three-eighths normal diameter.) Kidneys showing the characteristically coarse and irregular scarring found in certain cases of chronic pyelonephritis. Several calyces underlying depressed surface areas have an inflamed and very engorged mucous lining.

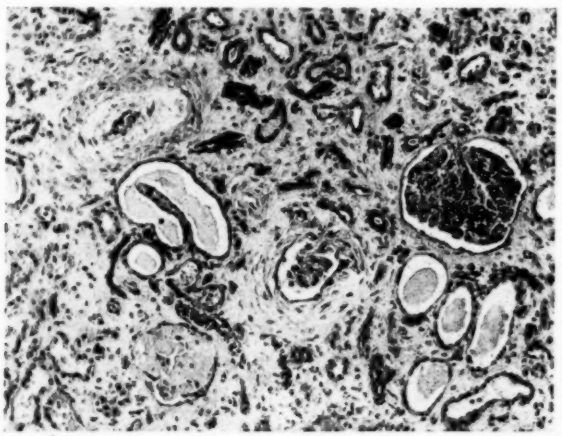

FIGURE X: Case IV. Chronic pyelonephritis. Advancing fibrosis, glomerular destruction and tubular atrophy in chronic pyelonephritis. Towards the left can be seen an almost obliterated interlobular arteriole. (×96.)

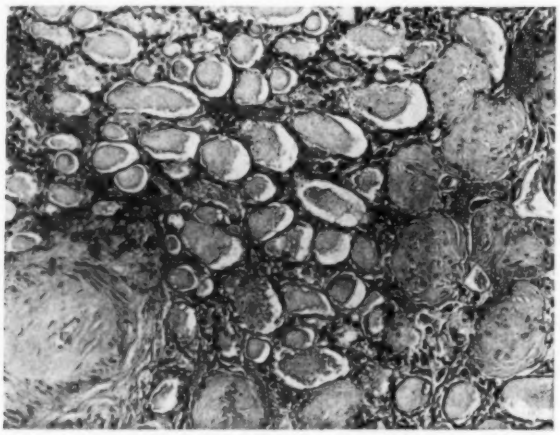

FIGURE XI: Case IV. Chronic pyelonephritis. Showing fibrosed glomeruli, an occluded interlobular arteriole and the thyreoid-like structure presented by the dilated tubules. (×96.)

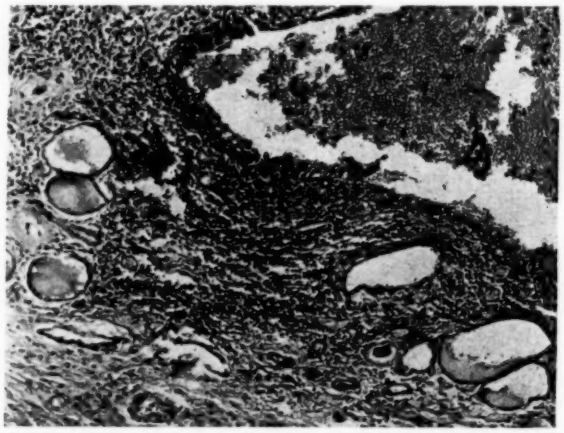

FIGURE XII: Case IV. Chronic pyelonephritis. The cavity of the renal pelvis contains effused blood. From the mucous surface the epithelium has been partly shed. Numerous inflammatory cells are present in the connective tissue of the renal pyramid, in which dilated collecting tubules with hyaline contents are to be seen. (×96.)

ILLUSTRATIONS TO THE ARTICLE BY Dr. A. J. CANNY.

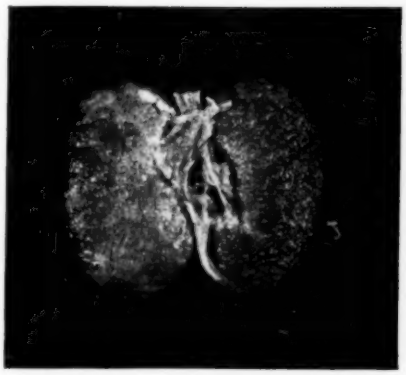

Figure XIII: Case V. Hypogenetic kidney (Fahr). (Reduced to half normal diameter.) This kidney is much smaller than the normal. The uniform granularity of moderate coarseness can be distinguished in that half of the surface from which the capsule has been removed.

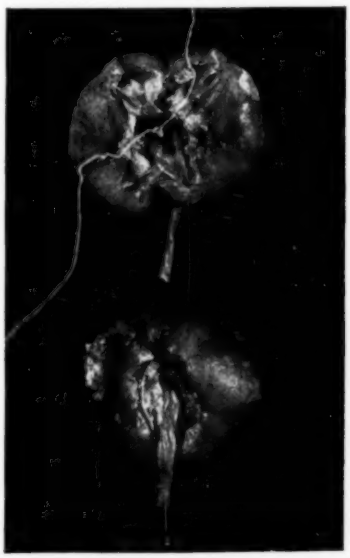

Figure XVI: Case V. Renal dwarfism. (Reduced to half normal diameter.) The surface of the kidney is smooth. There is no clear demarcation between cortex and medulla

ERRATUM.

Attention is drawn to the fact that Figure IV has inadvertently been printed upside down.—Editor.

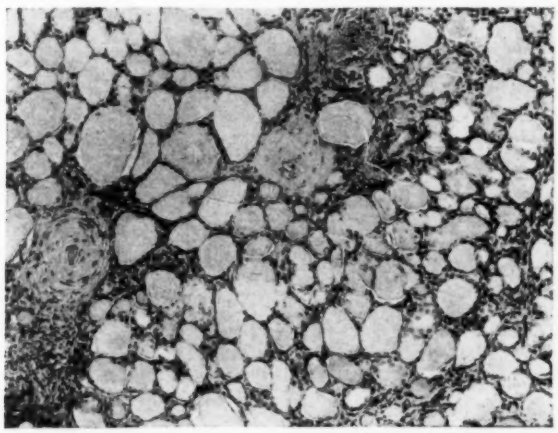

Figure XIV: Case V. Hypogenetic kidney (Fahr). The thyreoid-like appearance produced by the accumulation of hyaline material in dilated tubules is shown. Towards the left border of the illustration an obliterated interlobular arteriole is distinguishable. (× 93.)

Figure XV: Case V. Hypogenetic kidney (Fahr). Showing the fibrous extrapelvic connective tissue containing no tubular structures. In this tissue a few wandering cells, chiefly lymphocytes, can be distinguished. (× 81.)

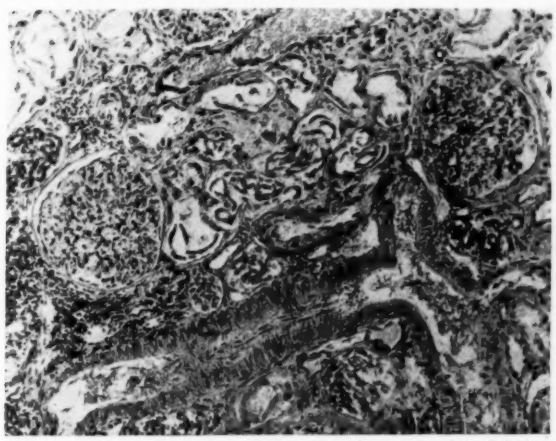

Figure XVII: Case VI. Renal dwarfism. An interlobular artery is shown, free from sclerotic lesions in the wall, and with a wide lumen, in which desquamated endothelial cells are present. (× 96.)

ILLUSTRATIONS TO THE ARTICLE BY DR. FRANK TIDSWELL.

FIGURE I.—Museum preparation showing early stage of oil infiltration near bronchi.

FIGURE II.—Museum preparation; late stage, showing extensive and extending consolidation.

ILLUSTRATIONS TO THE ARTICLE BY DR. KAYE SCOTT.

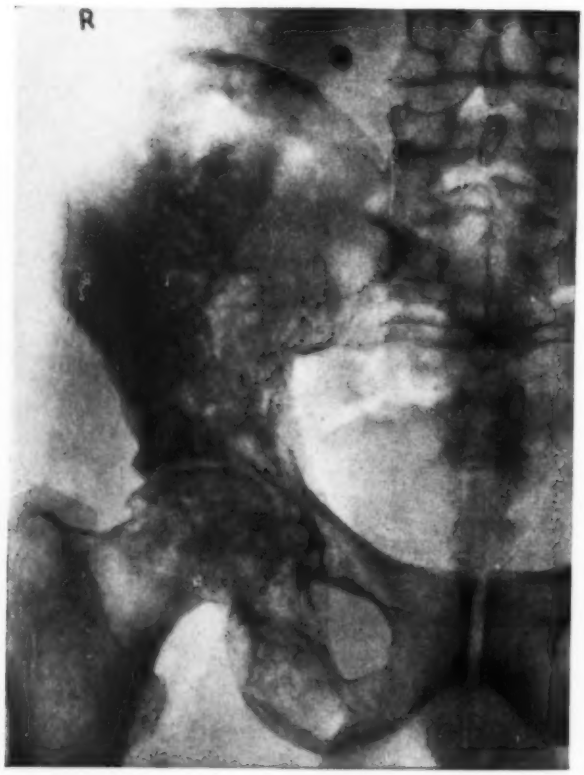

FIGURE I.
March 23, 1935. Osteoporosis of the right ischium and the lower portion of the right ilium due to hydatid disease.

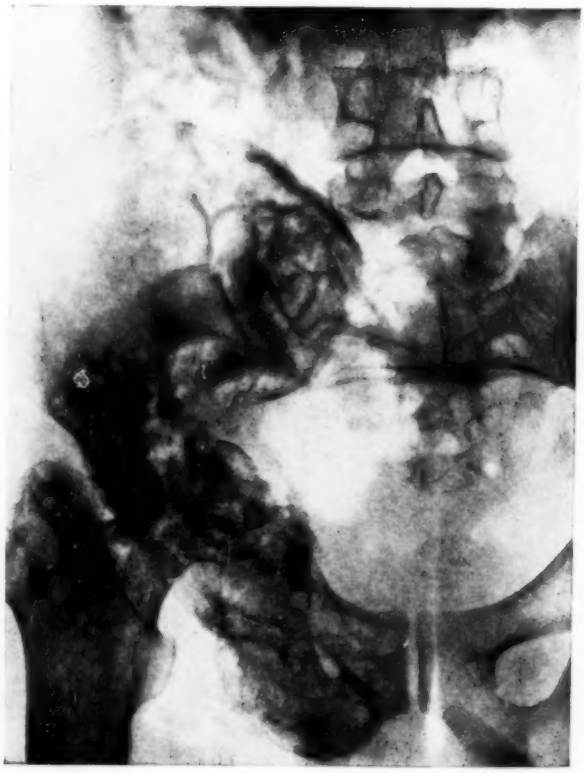

FIGURE II.
December 9, 1938. Further displacement of the acetabulum and further extension into the ilium towards the right sacro-iliac joint.

of each rib bed and the intercostal bundles are divided posteriorly and a piece of each one and a half to two inches long is removed. The nerves and arteries are secured as they join the bundles. Commencing posteriorly, the attachments of the pleura to the neck are gradually defined, ligated and divided. After the division of these structures, the dome of the pleura will contract down below the border of the fourth rib. The whole procedure of apicolysis takes anything from twenty to seventy minutes, depending on the firmness of the adhesions.

The wound is then closed by suture of the scapular muscles in two layers. The space over the apex is drained by a tube taken out through a stab wound in the axilla. Firm strapping is applied from the ribs on the unoperated side posteriorly under the axilla to the sternum anteriorly, and from the sternum anteriorly over the neck and down to the lower ribs posteriorly on the side of operation.

During the operation there may be injury to the first dorsal nerve where it crosses the neck of the first rib, injury to the brachial plexus or injury to the great vessels. All these happily are rare. The cervical sympathetic may be injured during the dissection, and Horner's syndrome may result. The cavity in the lung may be opened; this also is rare, but is serious if it does occur.

There is always some shock, and it may be severe. When there is severe shock the further drop in blood pressure entailed by the bandaging, turning and placing of the patient on a trolly may be fatal. When shock occurs during operation, blood transfusion should be given, and if at the end of the operation the blood pressure is still low, the patient should not be turned or moved until the pressure improves. The bed should be brought to the operating theatre, and the patient should be turned and put into position in bed all in one action.

The fall in blood pressure consequent on the turning and moving of the patient varies between 10 and 25 millimetres of mercury, and occurs whether the anæsthetic is continued or not and no patient whose systolic blood pressure is below 90 millimetres of mercury should be turned or moved. It follows that an essential for safety in operation is continuous observation of blood pressure, pulse and respiration.

There is a fair amount of hæmorrhage associated with the resection of the ribs, and it is one of the factors in the cause of shock and is treated by blood transfusion.

The space over the apex of the lung fills with fluid, some of which is drained away by the tube, which is removed after forty-eight hours. The fluid which remains keeps the apex of the lung down. At the second stage, when the fourth rib is removed, this space is often opened. It is found to be lined with a strong membrane, and may still contain a few ounces of blood-stained fluid. Very rarely the fluid in this space may become infected. If so, efficient drainage must be established, in which case healing may occur. Infection of the wound is relatively common. The infection is usually by pyogenic organisms and mild in degree. The source may be from the extrapleural glands or from the exterior, since the incision is large and open from one to two hours. Every precaution must be taken to prevent droplet infection by the use of efficient face masks, and to secure asepsis in the operation area. Severe infection may occur, and is, of course, a serious complication.

The middle stage is carried out through the lower part of the incision used for the upper stage. For the lower stage a vertical incision is made in line with the vertical portion of the incision, but not continuous with it. These stages differ from the upper only in the amount of rib removed. The posterior limits of the resection are the same, but the anterior limit is gradually reduced according to the amount of disease present. All the ribs overlying any area of disease and one below are removed. After each of these stages a firm pad is strapped over the operation area to provide temporary rigidity of the chest wall. This facilitates coughing and prevents paradoxical movement. The completion of each stage increases the amount of contraction possible in the preceding stage if the rib beds are still mobile. The intervals between the stages therefore should not be longer than three weeks, for in this time regeneration is well advanced in the beds of ribs already removed.

After any stage of thoracoplasty, but particularly after the upper stage, there are two grave complications; these are bronchopneumonia in the affected lung, and spread of disease to other parts of the lungs, usually to the middle zone of the other lung. The bronchopneumonia is always preceded by atelectasis. By making routine X-ray examination of all his patients after operation, Semb found that 50% had some degree of atelectasis.[7] This atelectasis is due either to the blocking of a bronchus by sputum or less commonly to kinking of a bronchus during the contraction of the lung. A diminution of sputum was always found in patients who had atelectasis, and often preceded it. Patients able to use their diaphragm were much less liable to atelectasis. Thus, of 56 patients with paralysis of half the diaphragm as a result of interruption of the phrenic nerve, 36, or 64%, developed collapse; while of 48 patients without diaphragmatic paralysis, 15 patients had atelectasis. Of these 15 patients, in 12 there was moderate or complete fixation of the diaphragm by adhesions. Moreover, out of 27 patients with a free diaphragm, only three had atelectasis, whereas of 64 with a fixed diaphragm, 43 had atelectasis. Thus there can be no doubt whatever that immobility of the diaphragm is a potent cause of collapse, and therefore in any patient with unilateral disease no permanent paralysis of the diaphragm should be carried out unless there is a contraindication to subsequent thoracoplasty.

The importance of atelectasis is that it may so reduce the vital capacity of the patient as to cause death from suffocation, and that it is a precursor of bronchopneumonia. Post-operative bronchopneumonia is at first a non-tuberculous bronchopneumonia, but a tuberculous infection soon supervenes. In fact, if the patient lives longer than two to three weeks, infection with tuberculosis is always present.

Spread of the disease after the operation is a very disappointing sequel. The conditions necessary for spread are lowered general and local resistance. Both of these are present to an extreme degree immediately after operation, and if there is any shock, there is, for all practical purposes, complete absence of general resistance, and it is in these cases that spread is most likely to occur. In every case I have seen in which spread occurred, severe shock had been a feature.

Heart failure occasionally occurs, and is diagnosed only after the death of a patient who has had a persistent tachycardia, and in whom no evidence of other cause of death is found at the post-mortem examination.

Paralysis of half of the diaphragm is brought about by crushing or evulsion of the phrenic nerve. With local anæsthesia, a transverse incision is made in the neck one inch above the clavicle over the posterior border of the sterno-mastoid muscle with the head turned to the opposite side. The sterno-mastoid muscle is retracted medially and the superficial surface of the *scalenus anterior* is exposed. The nerve lies in the fascia on the surface of this muscle. There is no difficulty in finding the nerve unless this fascia is disturbed. If the nerve is to be crushed, it is picked up and crushed by several applications of a clamp. If it is to be evulsed, it is cut after being picked up, and the distal end is grasped in a clamp. It is freed slightly by gauze dissection and withdrawn by steady pressure in the line of the nerve until it breaks. At least 10 to 12 centimetres of nerve should be secured to make certain of removing the connexions of any accessory branches.[9] The withdrawal of the nerve should be stopped at once, and the nerve cut, if there is any complaint of pain in the chest. A search should be made for accessory branches of the phrenic nerve.

Radical treatment of pulmonary tuberculosis by removal of the diseased area is possible only when that area is confined to the periphery of one lobe or one lung without any infection of the hilar structures.

Although the first successful pneumonectomy, that of Macewen, was performed for pulmonary tuberculosis, the method has not been developed as yet.[10] Such lobectomies as have been performed have had disappointing results. The severity of both operations is such that, should any diseased area be overlooked and left behind, rapid advance

will occur. The difficulty of avoiding this is almost insurmountable.

External drainage of very large cavities has also been suggested. It was probably first carried out in the fifteenth or sixteenth century, when one, Pheracus, mortally ill with a large cavity, placed himself in the forefront of battle. Here he received a sword thrust which entered his cavity. His courage met with due reward, for his disease cleared up after the drainage.[11] Anyone who has watched a patient with a very large cavity exhausting himself by raising 20 to 25 ounces of sputum daily, cannot but reflect that even if the wound became infected with tuberculosis, as surely it must, the beneficial effects of the relief of the cough as the pus drained into the dressing, would make external drainage worth while. Should the cavity then collapse before infection of the wound has occurred, there might even be considerable improvement in the condition of the patient. However, this is at best a desperate measure.[12]

Of the measures available in the management of tuberculosis, it is apparent that artificial pneumothorax allows complete concentric contraction of the lung. The collapse is gradual, and its production calls for little endurance on the part of the patient. It has, therefore, little effect on either general or local resistance.

Cauterization of adhesions causes little interference with the mechanics of respiration; but the sudden expansion of the lung, should the air be forced out of the pleural cavity by coughing immediately after the operation, may cause some disturbance of the diseased area. Some endurance is required during the operation, and should any serious complication such as infection occur, there may be considerable lowering of general resistance. However, the benefits of adequate collapse will more than compensate for any complication except infection of the pleural space.

Extrapleural division of adhesions, while it calls for considerable endurance on the part of the patient, results in very much less lowering of general or local resistance than thoracoplasty or the completion of extrapleural pneumothorax.

Extrapleural pneumothorax involves a sudden collapse of a large portion of the lung. The mechanical upset may be great, but usually can be tolerated. It involves moderate lowering of general resistance and probably rather more lowering of local resistance. Recovery of general resistance as indicated by the return of sedimentation rate to pre-operative levels is rapid. However, the incidence of complications is high. Overholt and Tubbs[25] report four cases of infection in 31 cases, and three cases in which they were unable to remove the blood-containing effusion. These were early complications. In those which I have observed, infection has occurred in three cases after several weeks. Roberts[14] had three deaths in 33 cases, and had to abandon the operation in three instances. Refills must be maintained as for an intrapleural pneumothorax, although after some weeks the frequency of the refills can be lessened by an introduction of oil at low pressure instead of air.[13] What the end result is to be when the disease is quiescent remains to be seen, for reexpansion after the cessation of fills must be a somewhat hazardous proceeding.

Thoracoplasty allows of excellent contraction of the lung, particularly when the Semb procedure is carried out. It produces a permanent change in the chest wall, and causes some deformity. It occasions great interference with mechanics of respiration in the post-operative period. Ventilation of the good lung is impaired as well as that of the diseased lung, until the rigidity of the chest wall is restored. The paradoxical movement may cause sputum to be aspirated throughout the lungs. There is a very considerable lowering of general and local resistance. The resistance is indeed almost negligible for a time, varying with the degree of shock from one to twenty-four hours, and it remains at a low level so long as any reaction persists. It follows that only patients with considerable reserve of vital capacity and good powers of compensation, and whose general and local resistance are high, can withstand the strain of the operation and survive the post-operative period without serious complications. Despite the disadvantages, it offers to these patients a good chance of the benefits of collapse therapy, with a return to work for over one-third of them.

Hedblom and Van Hazel[26] collected 3,762 cases of thoracoplasty without apicolysis and added 200 of their own. Of these, 236 were partial thoracoplasties only. The mortality rate over all was 10·3%. A follow-up for periods from one to twelve years showed 35% of patients to be symptom-free and at work, 22% improved and able to do some work, 5% worse, 33·6% dead, and 3·5% untraced.

The results of thoracoplasty with extrafascial apicolysis in Semb's hands were as follows: of 246 patients, 6% died within two months, 5% died later and 219 survived. Of these, 198 or 90% had complete collapse of the cavity as shown by skiagram, and 108 or 86% had sputum in which tubercle bacilli could not be detected. When the operation was carried out in stages, the mortality rate, early and late, was reduced from 11% to 6%. When the upper six ribs were removed in one stage, 20% of patients had a severe reaction, 25% had atelectasis and 4% died within two months. When more than six ribs were removed, the figures were 21%, 62% and 16% respectively; when the upper three ribs only were removed, the figures were 6%, 4% and 2%.[17] In an earlier series of 104 cases, in 21% severe shock occurred, in 20% there was some wound infection, but in only 5% was it severe; there were nine cases of bronchopneumonia, of which five were fatal; in all nine atelectasis was present prior to the bronchopneumonia; there were five cases of suffocation due to obstruction of a main bronchus by pus and mucus.[7]

Paralysis of half the diaphragm reduces the capacity of that side of the chest to a small degree. It also reduces the amount of movement of the lung on the same side. Although its effect on the vital capacity is slight, if this is already low either from extensive disease or from any cardio-vascular defect, the result of loss of the diaphragmatic movements even on one side may be disastrous.[18]

Clinically, patients suffering from pulmonary tuberculosis may be seen when their resistance is low or decreasing, or when it is high or increasing. The first group is characterized by pyrexia, increasing amounts of sputum, hæmoptysis, loss of weight, lassitude, acceleration of the blood sedimentation rate, and the appearance of new areas of disease and "soft" areas of infiltration in serial skiagrams. In the second group there is no pyrexia, the amount of sputum is constant, there is no history of recent hæmoptysis, the weight is constant, there is no lassitude, the blood sedimentation rate is little faster than normal, and skiagrams show "hard" areas of infiltration and no evidence of new disease. A third group consists of patients between these extremes.

The first step in the use of collapse therapy is to attempt an artificial pneumothorax, with the exception that when the disease is confined to one small area in the lung, with a cavity under one inch in diameter, temporary diaphragmatic paralysis is tried. If this measure is successful in controlling the disease, it can be made permanent.

If artificial pneumothorax is impossible for patients in the first group, only diaphragmatic paralysis is available. Temporary paralysis is indicated always in this group unless there is already some contraindication to thoracoplasty.

Patients in the second group, who have fair vital capacity and whose ribs are sufficiently resilient to compensate for the new positions consequent on the operation, are suitable for thoracoplasty. That is, there must be the equivalent of at least one whole lung unaffected by disease, the ribs must not be calcified, the age should be below fifty years, and no emphysema or cardio-vascular defect should be present. For patients in the second group unsuitable for thoracoplasty, and for those in the third group, extrapleural pneumothorax may be considered.

Diaphragmatic paralysis is also indicated when artificial pneumothorax therapy is terminated, to lessen the amount of expansion and the use of the lung.

Thoracoplasty is indicated in pulmonary tuberculosis complicated by empyema.

Thus the surgical treatment of pulmonary tuberculosis is the surgical closure of cavities in the lung. None of the procedures is without danger, and the major operations have a considerable mortality rate. On the other hand, they offer a possibility of a return to work and a definite prolongation of life. So long as a cavity is open, death is certain, except in the aged, within a few years. No amount of surgical treatment can replace the measures to deal with the general resistance, of which the chief one is rest to the body as a whole, rest in congenial surroundings on a nutritious diet. Success can be won only by the closest cooperation of physician, surgeon and patient.

The proper procedure to be adopted in any given case can be selected only when the exact state of the patient and his disease is known. As Lilienthal[19] states:

Just as in mathematics, there must be a knowledge of the position of at least three points to determine the character of a curve, so in tuberculosis it requires two, or better, three observations to appreciate the progress of a case so as to choose to advantage the time and the procedure which will promise the best result. It is thus seen that if a decision to operate is to be made at any particular period, a knowledge of the condition present must be rounded out by the report of intelligent observations made in the same case during the preceding months or years, and preferably from its incipience.

To surgery in the treatment of pulmonary tuberculosis and its pioneers, John B. Murphy, Sauerbruch, Morriston Davies, Roberts, Lilienthal and Graham, Osler's words may well be applied:[20]

What has been accomplished is only an earnest of what shall be done in the future. Upon our heels a fresh perfection must travel, born of us, fated to excel us. We have but served, and have but seen a beginning.

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CONTROL OF TUBERCULOSIS.[1]

By DARCY R. W. COWAN, M.B., B.S., F.R.A.C.P.,
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A DISCUSSION on the control of tuberculosis is not out of place in war time, as experience has shown that it is just at such times of stress and privation that tuberculosis makes headway. One shudders to think what is likely to befall the half-starved peoples of Europe who have fallen under the German yoke. Australia has not yet suffered the ravages of war on her own territory; but at least we remember the aftermath of tuberculosis among our soldiers who fought in the last war. Owing to the wise precautions that have been taken on this occasion to eliminate the obviously tuberculous at the outset, the toll is not likely to be so great. But he would be a super-optimist who thought there would be none. It is almost certain that some men who appear to have overcome their primary infection will suffer manifestations of reinfection as a result of the hardships to which they are likely to be subjected.

The control of tuberculosis in this State is a matter of some importance. Quite apart from the Repatriation Department, of which I have no figures, there are about 1,000 persons receiving an invalid pension as a result of this disease; this represents an annual outlay by the Commonwealth Government of approximately £50,000. The State control costs almost as much. In addition to this, we are losing citizens at the rate of nearly 200 a year from this disease. At a moderate valuation of £500 a head, this makes a further loss of £100,000—a total of about £200,000 a year. Also we must not forget the enormous toll in human suffering. In the past thirty years, well over 100,000 people in Australia have died from tuberculosis, a preventible disease. This state of affairs constitutes a challenge to the medical profession, and a test of the statesmanship of our democratic institutions. It always puzzles me to know why almost unlimited money is available for defence against the enemy outside our gates, while there is the greatest difficulty in securing supplies for a concerted attack on the more insidious, but none the less dangerous, enemy within. Dr. Cumpston has quoted the late Victor Heiser of the Rockefeller Foundation as saying that we should have no tuberculosis in Australia. Heiser was right; but what are we doing about it?

I might perhaps tonight have confined myself to a few polite platitudes, but I have decided to speak of things as I see them in the hope that progress may result. It is not that I have anything new to say. The late Dr. Ramsay Smith spoke in much the same strain over thirty years ago. The Commonwealth and State health officials know about these matters quite well, but appear impotent to get anything done. No doubt they have directed the attention of their ministers to what is needed, and who can blame them for not making a fuss? Under our present system, that is no way to get along in the Government service; and naturally these men must think of their jobs. It may be objected that we are not a political body, and should not touch on political aspects of this problem. Certainly we are not a political body, and that, I submit, is all the more reason why we should attempt to enlighten the politician, and if necessary the public as well, on the serious nature of this problem and the measures necessary for its control. In this State, the control of tuberculosis is in the main a Government activity, and I cannot contribute much of value in this paper unless I touch on some aspects of its administration.

The control of tuberculosis in theory is easy. All that is necessary is to discover those persons who have "open" lesions, to isolate them during the whole of the infectious stages, and to keep on doing this. But in practice it is not so easy. A survey of the whole community is a practicable procedure, and should not be unduly expensive; but the isolation of all infectious patients is impossible. I

[1] Read at a meeting of the South Australian Branch of the British Medical Association on September 26, 1940.

am not opposed to a survey of the whole community; but I think it is essential, before such an ambitious programme is embarked upon, that definite provision should be made beforehand for the care, both medical and economic, of sufferers who may be found. The control of tuberculosis may be considered conveniently under four headings: (i) prevention, (ii) "case finding", (iii) treatment, (iv) after-care. Dr. Wunderly has had special experience in "case finding" surveys, and he will deal more especially with this aspect of the problem. I do not propose to repeat my presidential address of 1936, to which members have access, but to confine myself to certain aspects of the problem which seem to me of especial importance.

Though I am no pessimist in regard to treatment, yet in the main I feel that we must agree with the conclusions of D'Arcy Hart, who in his Milroy Lectures for 1937, published in *The Lancet*, showed that, despite all modern methods of treatment, the prognosis for the individual patient with established disease had shown little, if any, improvement. It is quite certain that many patients come to us in such an advanced stage of the disease that cure is out of the question, and all we can do is to secure some measure of relief for these patients and prolong their lives. And so, prevention and early "case finding" must come into the forefront of the campaign. Prevention is the particular function of the boards of health, but they can do little without the whole-hearted support of every practising member of the profession. Complete segregation of all patients through the whole of the infective stage of the disease is impracticable; but it should be attempted through the more acute and more infectious stages. Recently I saw a man, aged seventy-five years, who had had a hæmorrhage from the lungs at the age of twenty-five years, and who had been intermittently infective through the whole of the intervening time. You cannot isolate a man for fifty years! The only practical alternatives are partial segregation and education in preventive measures. I have at times criticized the health authorities, and I shall probably do so again. But I do very willingly acknowledge the good work they are doing under conditions that are far from ideal. As a practising physician, I feel that it is unwise to burden the patient with too many instructions. If we could concentrate on the two or three things that are important and not worry about the multitude of things that do not matter much, I feel that we should receive more cooperation from the patient, and so achieve better results. Dr. Ramsay Smith laid down the rule that "infection comes in through the mouth and goes out through the mouth". If to this we added "and the nose" we should get at the crux of the problem. This entails collection and disposal of sputum, and care in coughing and sneezing. I believe that unguarded coughing is the greatest danger. It is seldom that a person will spit into another's face, but he will cough into it with impunity. Droplet infection is a real danger. The mouth and nose should be completely covered with a handkerchief every time the patient coughs or sneezes. Personal cleanliness is essential, and especially care with handkerchiefs. In a reasonable household, the danger of infection from feeding utensils is negligible. But the old fetish continues. Nothing worries the average patient more than having to use marked feeding utensils. This is not a highly contagious disease, and a careful, cleanly person carries little risk of infection to others.

On more than one occasion recently privately controlled hospitals have evaded or attempted to evade their moral responsibility in the matter of providing accommodation for nurses who have contracted tuberculosis while in their employ. The reason given is fear of infecting others. I submit that a hospital management which doubts its ability safely to accommodate a patient suffering from pulmonary tuberculosis has no right to a licence to conduct a hospital. And a hospital which turns out its nurses in total ignorance of the nursing requirements of this common disease has no claim to be registered as a training school for nurses. It is deplorable that hospital managements should pander to public fears by entertaining such outrageous ideas. If these patients are not safe in a properly conducted hospital, then where are they safe? It is, of course, the unrecognized case, in which no precautions are taken, that is the greatest source of danger. This fact is constantly being forced upon our notice in the examination of "contacts" at the Chest Clinic. Here is a typical example:

> The father had a cough for three years, for which he had been medically examined. But the cause of his trouble was not recognized until he was within a short time of death. His wife has been examined and found to be infected and to have an early apical lesion. The baby, aged eleven months, also reacts to the Mantoux test.

An early diagnosis is essential if success is to be attained in the prevention and control of this disease, and in this matter the practitioner must play an important part.

One of the great danger periods in tuberculosis is the primary infection stage in young children. Figures suggest that from 7% to 10% of children who become infected before the age of two years die of meningitis or other generalized infection. An ever-present problem is the management of young children in tuberculous families. The obvious thing to do would seem to be to remove the children from the infective environment, at any rate for a few months at a time. Sometimes the infecting patient can be removed, but not always. A "preventorium" to which the children can be sent is almost a necessity. The Government has been offered as a gift an excellent home for such a purpose, but is reluctant to accept because it will cost a few hundred pounds a year to run. I can see now a young mother with flushed cheeks and a husky voice bringing in two small children for examination. Contact with the infective mother is dangerous for the children, and the burden of caring for the children is too much for the mother. The establishment of a "preventorium" is a practical measure in prevention which would pay handsomely.

I want to touch briefly on "case finding". If you read "The Diary of a Doctor" you will learn that in the average case tuberculosis is a symptomless disease for the first two years. If you see *Pix*, you will remember the slogan: "Tuberculosis is seen, not heard." If you study the literature, you will recall the famous remark concerning the stethoscope made many years ago by Rivière.

More recently Hurn said: "I believe that on balance more good than harm would result if physical examination of the chest was prohibited entirely in the diagnosis of pulmonary tuberculosis." My sympathy is with the practitioner; but I would ask him to remember that there is such a disease as pulmonary tuberculosis, and that it is almost impossible to diagnose it in the early stages by ordinary methods of examination. There is less excuse at the present time when facilities for diagnosis, free of charge, are available at the Chest Clinic for all, irrespective of their financial position. This is a very valuable concession which the Government is providing, and I have no doubt that its scope will be extended as circumstances permit. I know from personal experience the difficulties of inducing "contacts" who feel well to submit to special examinations, which may cost them two or three guineas. Here is the record of a family, of which the first member to appear was a young man affected with a rapidly advancing pulmonary tuberculosis:

> The father was advised that the young man should be put into an institution. But he insisted on taking him home, where his three sisters lived. The risks were explained and he was advised that the girls should be examined; but he neglected this advice, as the girls seemed well. The boy died three months later. Within two years the first girl appeared at the clinic. She had active tuberculosis, from which she died. In the same month the second girl appeared at the clinic. She had active tuberculosis, from which she died. Shortly after, the third girl appeared. She had active tuberculosis, with which she has been struggling ever since.

We want to avoid this sort of tragedy. How much better it would have been to recognize these cases in the preclinical stage, when treatment would have offered a reasonable change of arrest!

I have heard it said that it was a mistake to make special examinations of soldiers for tuberculosis. I do not subscribe to this opinion, and I shall now show you X-ray films of a nurse and several soldiers who passed their ordinary

medical examinations but were rejected as the result of X-ray examination.[1] Each of these had active tuberculosis, and tubercle bacilli were demonstrated in the sputum in every case but one. If they had been accepted for service, the danger to themselves and to others is obvious; and the pensions aspect also is important. The outstanding feature of all of these cases is the extent of the damage to the lungs as disclosed by the radiograph, which is in such pronounced contrast with the paucity or absence of symptoms and clinical signs. If only one subject of active tuberculosis had been recognized in each 1,000 persons examined, the examinations would have been worth while. But Dr. Wunderly will tell you that one or more was found in every 100 examined.

One of the problems that has come before us rather acutely as a result of these surveys and of the examination of contacts is the difficulty of assessing whether a lesion is an active one that needs treatment or merely a healed one of no particular clinical importance. This is by no means easy, and the radiologist is no more infallible than anyone else in this regard.

The radiologist's report on one patient was as follows: "Healed and calcified lesion at the left apex." But the patient was coughing up sputum containing tubercle bacilli!

In another instance the patient seemed quite well and the lesion was found on routine examination. In the radiograph it has the appearance of a well-defined area of fibrosis at the apex. The patient was put to bed for a month, during which time she had no symptoms at all, her temperature did not once rise above normal, and her blood sedimentation rate was normal. A second X-ray examination at the end of a month revealed no change. The lesion was looked upon as healed, and the woman was allowed to go back to work. Within a fortnight she caught a "cold", with cough and sputum, in which tubercle bacilli were demonstrated. With rest in bed the symptoms quickly subsided. She took a trip to England, where she went through a winter and did hard work as an ambulance driver and in other ways. She has had no recurrence of symptoms, and X-ray examination still reveals no change.

Caution is needed in such cases, and very careful clinical observation. We should not hurriedly conclude that a lesion is inactive. Careful periodical examination may be the only way of arriving at a conclusion, and the radiologist must have a second chance just the same as anyone else. Many of the rejects from the Australian Imperial Force come in this category.

A young man, whose only illness has been a mild attack of influenza, is very fit and played Rugby football during the past winter. In the X-ray film are seen tiny scars at the apices of both lungs, almost certainly the result of a tuberculous process. Careful observation over a month failed to detect any evidence of activity. He was rejected from the Australian Imperial Force.

Another man had a spontaneous pneumothorax five years ago, and his father and brother died of tuberculosis. He has small scars at one apex, but he was accepted. The appearances in an X-ray film after he had spent three months in the Woodside camp strongly suggest an extension of the tuberculous process.

These cases seem to me to be hardly consistent. If only unexceptionable lives are wanted, why accept a man who has a bad family history and has had a spontaneous pneumothorax and reject another with almost similar X-ray findings, who has had neither of these things? I am afraid that the pendulum has swung altogether too far, and that we are in danger of allowing our clinical acumen and sound judgement to be unduly influenced by some minor or doubtful X-ray findings.

A man was rejected from the militia because of the appearances in an X-ray film. He comes from a country centre, where they want him badly for clerical work, at which he is expert. He is aged about forty-five years, says he never felt better, and plays strenuous tennis *et cetera*. A small circumscribed area of opacity, about the size of a threepenny piece, is seen in the left lung field near the periphery. I am willing to take a chance that this is a healed primary focus, and I have written to the local centre that there is no evidence of active tuberculosis; but I should like to examine him radiologically again in six months' time.

[1] At this stage Dr. Cowan showed some X-ray films.

There are limits to the value of radiography; it cannot tell us everything. As a result of the absence of radiographic evidence after a spontaneous pneumothorax or a pleural effusion has cleared up, it is being taught that these conditions are not dependent on tuberculous infection. It may be that some of them are not tuberculous in origin, but the majority are. I shall believe the contention that these cases are relatively seldom tuberculosis when someone brings forward a series of cases in which not only is the X-ray film clear, but the Mantoux test produces no reaction. There is only one safe rule with such conditions and that is to look upon them as tuberculous unless it can be proved they are not; and the only way to do this with any degree of certainty is by the Mantoux test. And it is well to remember that allergy may take a few weeks to develop, so it is a wise precaution to repeat the test a few weeks later in the absence of a reaction, and to use a 1/100 dilution. The surest test of tuberculous infection is the tuberculin test, just as the surest evidence of disease usually comes from the radiograph. But the radiologist, like everyone else, cannot tell when a tuberculous infection will be overcome without the development of actual disease. All he can possibly say is that, in a particular case, evidence of disease has developed, and he cannot in all cases say with certainty what that disease is and whether it is an active process.

I am fully aware of the great value of the X-ray film in diagnosis, but interpretation is not always easy.

In two of my cases the diagnosis of acute tuberculous bronchopneumonia was made. In one the prognosis was given as hopeless. But neither patient was tuberculous, and both made a complete and uneventful recovery.

The radiologist cannot, and he should be expected to, make a diagnosis between tuberculous and non-tuberculous broncho-pneumonia. If the lesion is apical, and especially if cavitation is present, he is on safer ground. But even here he must be careful.

In one case the X-ray film showed patchy consolidation with cavitation of the upper lobe of the right lung. It had all the appearance of a tuberculous lesion. Actually it was a lung abscess with surrounding inflammation, caused by the inhalation of a fragment of tooth, and it cleared up completely without any special treatment.

Infection of the upper air passages associated with a chronic cough is often overlooked or wrongly diagnosed.

A short time ago a patient was sent from the country for admission to the Morris Hospital. She had had a cough for years, but it was not due to tuberculosis. She has antral infection with chronic bronchitis.

Another was sent into Frome Ward for termination of pregnancy. She had been looked upon as tuberculous for years. But she was suffering from bronchiectasis in association with antral infection.

In one week I saw three boys sent for examination for coughs persisting over years. Their mothers had been told that "it was nothing, they would grow out of it". None reacted to the Mantoux test and all had chronic antral infection.

The fact is, they will not grow out of it; they will grow into it and develop chronic bronchitis or bronchiectasis if their antral infection is not dealt with.

This case illustrates the late results:

The patient is a woman, aged twenty-four years, and she was rejected as a munitions worker on account of a chronic cough. She states that this started from an attack of pertussis when she was ten months old. She has been receiving medical attention for years, but no one had recognized the antral infection, and now she has well-established bronchiectasis.

I have time to say little with regard to treatment. Our armamentarium is limited and not very effective. It is the intelligent use of our weapons that counts. The treatment of tuberculosis, as of most other chronic diseases, is an art just as much as it is a science. Psychological aspects are too often overlooked. I would ask the practitioner to remember that there is more in treatment than masterly inactivity. The general hygienic dietetic plan must be the basis of all treatment; but there are other measures that are helpful in certain cases, such as collapse therapy in some form, tuberculin, and the administration of gold

salts. Before any particular line of treatment is adopted, it is necessary to have an X-ray examination of the lungs to ascertain the exact site and distribution of the lesions, and accurate clinical observations to assess the degree of activity of the disease process. The practitioner should know the indications for inducing an artificial pneumothorax, and should not deny the patient the benefit of this procedure, which is often life-saving.

Surely the old days are past when the patient was told: "You have tuberculosis. Go north."

I could tell you some lurid stories of this. I know of no specific healing virtues of the northern climate, and often the living conditions are quite unsuitable. Commonsense is desirable in the management of tuberculous patients. In a certain sanatorium a small billiard table contributed a little harmless relaxation for the patients. After a game one of the patients had a slight hæmoptysis. On account of this the billiard table was done away with. I asked an official if hæmoptysis occurred frequently, and what the patients were doing at such times. I was told that it occurred quite often, and almost always the patient was in bed at the time. Surprise was expressed when I suggested that going to bed seemed to be a dangerous procedure, and they should do away with all the beds in the sanatorium. Actually, all that was wrong with the billiard room was the fact that it was used also as a smoking room. It is curious that cigarette smoking, which I look upon as distinctly harmful to these patients, is not discouraged. But if one suggests a glass of ale—probably the best and cheapest stimulant for a jaded appetite—the management almost goes into a frenzy. Ale is not interdicted in Frome Ward, which fact possibly accounts for the patients' reluctance to leave there. I have never seen it do any harm; but of course if the patient wants a short life and a gay one, probably as good a way as any would be to drown his sorrow in alcohol.

Of special interest is the matter of the education of the medical student in tuberculosis. A few years ago the South Australian Branch Council of this Association had the temerity to suggest to the University of Adelaide that some improvement might be effected in this respect. The result was surprising. About this time a special department for patients with pulmonary tuberculosis was instituted at the hospital. For the next four years almost no students attended for instruction in the department. For four years the university turned out medical graduates who had had no practical clinical instruction in the diagnosis and treatment of this common disease. Diligent inquiries brought to light the interesting fact that this was solely the fault of the student. It was reported that he simply would not attend. Happily this is now a thing of the past. Definite arrangements have been made and students are showing no undue disinclination to attend.

Despite our best endeavours, only too often patients fail to make a complete recovery. They are left partially incapacitated, and in an infective condition. The only alternatives are to spend their lives in an institution, exist on an invalid pension, or attempt to resume their ordinary avocations, with the result that in many cases they break down again. The establishment of an industrial colony is the obvious solution of this difficulty, and some day, maybe, the authorities will realize that not only is this good for the patient and a safeguard to the health of the community, but it is a paying proposition for the State. If only twenty patients were taken out of institutions and made self-supporting, that fact would effect a saving of £3,000 a year to the State Government, to which amount can be added the saving to the Commonwealth Government in pensions. An industrial colony for partly incapacitated tuberculous patients is not an experiment or a new departure. Experiences in England at Papworth and Preston Hall have shown beyond all shadow of doubt the value of such schemes. Unfortunately our authorities are slow to move, and appear afraid that they will do something wrong. In an attempt to tighten up our public health administration, this Association recommended among other things the formation of a tuberculosis advisory committee to which it should have the right to nominate one member. The Government formed such a council, to which it appointed all three members; and perhaps one may be permitted to express mild surprise that two of its members should have no claim to any special knowledge of the subject. Some of us rather optimistically hoped that the council would be a live body with some constructive ideas. But our hopes were doomed to disappointment. In some respects it rather seems to obstruct progress. The problem of the patient with chronic infectious tuberculosis is a pressing one. It is wrong to force these men back into industry at a risk to their own health and to that of their fellow workers; but there is no reason for them to be scrapped. Given the chance, they can become useful members of the community. Early last year the Government stated that it would start a colony for convalescent consumptives, but nothing was done. Early this year a concrete scheme, with substantial backing, was placed before the Government. This scheme was rejected at the instance of the Tuberculosis Advisory Council. No one expects the Government to accept every scheme that is placed before it, and there can be no complaint because this particular plan was not adopted. But in considering it, the Advisory Council agreed to obtain, for purposes of comparison, plans and estimates of costs of an alternative scheme on similar lines. These plans and estimates were never obtained, and for this the Council cannot expect to escape criticism.

It is curious what difficulties one mets in trying to have some scheme of this sort started. It was first mooted in connexion with a sanatorium. After a long discussion, the Board seemed agreeable, but, before committing itself, decided to approach the Factories Department to see if there was anything in the act that might influence the position. It was somewhat of a shock to learn a few days later that no further action was to be taken for the reason that the Chief Inspector of Factories had advised that work of the sort proposed was detrimental to the health of the patients!

The economic factor in tuberculosis is of paramount importance. The plight of the family whose breadwinner is stricken with tuberculosis is tragic. The Federal Council of the British Medical Association in Australia and this Branch have made valiant efforts to secure some recognition of the needs of such families. Though some concession has been obtained from the State Government, the position in regard to pensions is still far from satisfactory. Governments fail to realize that this is an infectious disease. If the sufferer has to give up his work as much for the public safety as for his own, then the community should see to it that he and his family receive an adequate measure of relief. Through economic stress persons in an infective state are being forced back into industry. How is it possible to obtain any degree of effective control when conditions like this prevail? The pensions system in regard to tuberculosis is badly in need of overhaul. But the politicians will not look at it, and their responsible medical advisers become resigned.

Dr. Cotter Harvey has aptly described the person who "can't afford to be a case of early tuberculosis". I have seen dozens of such persons. They cannot afford to give up their work, and they carry on until they break down hopelessly. The *Pensions Act* lays it down that a person is not entitled to a pension until he is completely and permanently incapacitated. In the early stages, when a little financial assistance might result in the arrest of the disease, it is denied to the patient. It is given to him only when he is "down and out". There is no sense in this. In the case of the breadwinner with a family dependent on him, the assistance given is inadequate. If he goes to a sanatorium, which he should do in most cases for the sake of his family if for nothing else, the Government takes 14s. a week from the pension. The remaining 6s. will not pay the rent, so the patient goes home, to the danger of himself and the family. The family goes on "rations", and it is none too generous a ration for such people, who need extra nourishment to build up their resistance to the infection. The sufferer, because he is in receipt of a pension, is not entitled to further relief, and has to live on what is left over from the family's rations. As a result of the representations of this Branch some

concession has been gained, and on special recommendation these families are now allowed extra cash payments to cover rent and other incidental expenses. But the Relief Department is still inclined to quibble over details.

Then the position arises that patients whose disease has become arrested are dismissed by their employers, mostly through fear of infection. I have made it a rule not to encourage sufferers to return to work until it is safe both for themselves and for their fellow workers—in other words, until they are well and no longer infectious. But, despite a certificate to this effect, employers sometimes dismiss them. I have had some lively encounters over this, and I shall fight every time I see such injustices done.

Here is the record of a man, a returned soldier, who was treated nearly six years ago for pulmonary and laryngeal tuberculosis. He did well, and after six months' treatment the disease appeared to be arrested, and has been ever since. He was given a certificate that he was fit to return to work and was not infectious. His employers would not take him back and he has not been able to get work since. He receives a war service pension of £2 2s. a week. The house rental is 25s. a week, and with his wife and two boys he lives on 17s. a week. I asked him whether he would care to work in an industrial colony, where he might have a small house for about 13s. and a chance to earn a couple of pounds a week. He would jump at the chance; but no one will give it to him.

A properly conducted colony would almost pay its way; it would give a chance to many men and women to become self-supporting and self-reliant citizens again; it would effect a partial segregation of many infectious patients, with consequent protection of the community. But when a concrete plan is formulated it is "turned down cold" and nothing more is done.

Summary.

To summarize the requirements of the tuberculosis organization in this State, I would suggest the following measures:

1. The appointment of a Director of Tuberculosis, whose sole function should be to organize and direct the campaign. He should be freed as far as possible from routine clinical duties. No matter how good a man is appointed, his efforts will be unavailing if he does not receive the sympathetic and understanding support of the Government.
2. Improved facilities for "case finding" surveys. More and better clinics are needed.
3. Provision for after-care, especially an industrial colony for partly incapacitated patients who are capable of work under sheltered conditions.
4. A "preventorium" for children of tuberculous families.
5. A more rational scheme for economic assistance to sufferers from tuberculosis and their families.
6. A properly organized system of social service with a trained almoner at its head.

The aim should be a well-balanced scheme, in which arrangements, both medical and economic, for the care of patients with established lesions should go hand in hand with systematic "case-finding" efforts; and the preventive aspect, of course, should not be overlooked.

SOME UNUSUAL RENAL LESIONS ASSOCIATED WITH VASCULAR HYPERTENSION.

By A. J. CANNY.

(From the Department of Pathology, the University of Sydney.)

ON December 13, 1826, there was admitted to Guy's Hospital one William Bonham, who, though following the lowly calling of a cheesemonger's carter, was a sufferer from gout. This man, then in his fifty-fifth year, while not in his entirety an historical figure, was the possessor of organs the historic significance of which cannot be gainsaid. When he died on December 19, Richard Bright (1827) observed that his kidneys were granular, hard and smaller than the normal, and that his heart was "remarkably enlarged". Continuing his comments upon the heart of this man, Bright stated: "On the left side it was very thick and strong." Seeking for some relationship between the disorder of the kidneys and the enlargement of the heart, he was led to the conclusion that "the enlarged state of the heart would seen to bespeak some cause of obstruction to the circulation through the system beyond what we discovered".

This was the first occasion, so far as we have any record, on which some reciprocal relationship was suggested between sclerotic lesions of the kidney and an increased peripheral resistance in the vascular tree.

In his general remarks on the cases described in his well-known monograph, Bright (1827) commented that in some granular kidneys "when this disease has gone to its utmost it has appeared to terminate by producing a more decided alteration in the structure; some portion becoming consolidated so as to admit of very partial circulation". No suggestion was made that this ischæmic state of the kidney was in any way related to the obstruction to the circulation to which reference had previously been made.

Since that time controversies have raged as to the part played by diseases of the kidney in the production of arterial hypertension and as to the effect of increased arterial blood pressure on the structure and functions of the kidney. It is not the purpose of this paper to review or to comment upon the stupendous mass of literature, relevant and irrelevant, which accumulated during the century following the death of William Bonham. Five years after the centenary of the publication of Bright's reports, H. Goldblatt (1932) gave an account of the experimental production of vascular hypertension by the application to the renal arteries of dogs and monkeys of minute silver clamps, by the graded closure of which a greater or lesser diminution of the blood flow through the kidneys had been caused. Subsequent work by Goldblatt and his collaborators (1934, 1937) as well as by other workers, among whom Page (1935), Wood and Cash (1936), Glenn, Child and Heuer (1937), and Elaut (1936) are prominent, has established with reasonable certainty that ischæmia of the renal parenchyma leads to the formation within the kidney of a vasopressor substance which, by producing a more or less generalized vasoconstriction, raises the level of both the systolic and the diastolic blood pressure. It has been proved beyond reasonable doubt that no nervous reflex mechanism is involved in the production of this vascular spasm, but that the phenomenon is entirely of humoral origin.

If one renal artery alone is constricted, the blood pressure usually rises for some weeks or even for some months and then returns to a normal value; but when both renal arteries are compressed to a sufficient degree, yet insufficiently to cause any impairment of excretory function, there ensues a persistent hypertension.

In such diverse conditions as renal amyloidosis, hydronephrosis, polycystic disease of the kidneys, chronic pyelonephritis, renal hypoplasia, atheroma of the main renal arteries and *polyarteritis nodosa*, vascular hypertension is not uncommonly to be found even when the patient is far below the age at which hypertensive vascular disease might be expected and when the kidneys are free from the sclerotic changes in the small arterioles which are so characteristically seen in essential hypertension. Up to the present time little systematic attempt has been made to correlate the occurrence of increased systemic blood pressure with obstruction to the flow of blood through the vascular tree of the kidneys; but during recent years clinical evidence has accumulated which indicates that renal ischæmia may be the ultimate cause of certain cases of vascular hypertension in man.

Bell and Pedersen stated in 1930 that up to that time there was no record of pyelonephritis being associated with vascular hypertension. Some years later Longcope and Winkenwerder (1933) reported nine cases of chronic pyelonephritis associated with contracted kidneys. In three patients, the oldest of whom was aged twenty-eight years,

vascular hypertension had been present. Autopsy was performed in two of these three cases, but in neither instance is any information given about the structural condition of the renal blood vessels. More recently Longcope (1937) reported that in 11 of 22 cases of chronic pyelonephritis hypertension was present. Autopsy in three cases in which the hypertension was pronounced revealed only minimal sclerotic changes in the renal arterioles. No comment is made about the condition of the larger vessels, and as Longcope refers specifically to the absence or presence of only minimal hyaline sclerosis, one must assume that his remarks apply only to the afferent glomerular arterioles. He makes no reference to the presence or absence of intimal or medial thickening of fibrocellular or fibrous type which is seen so commonly in the kidney vessels in chronic pyelonephritis [Staemmler and Dopheide (1930, 1938), Fahr (1938)], and in the absence of any illustrations one is left uninformed as to the anatomical criteria on which a diagnosis of "chronic" pyelonephritis was based. Surely in the chronic phase of this disease fibrotic lesions must have been present in the kidney, and if so, the vessels in these areas must have exhibited some sclerotic changes. Longcope (1937) hints at the possibility of pyelonephritic hypertension being related to renal ischæmia, but seems to disregard the lesions in vessels above arteriolar size as a competent cause of such ischæmia.

Butler (1931) observed six children in whom vascular hypertension accompanied chronic pyelonepnritis. In two of these the renal disease was unilateral, and after the infected kidney had been removed the hypertension disappeared. A little later Barker and Walters (1938) also reported success in the treatment of vascular hypertension associated with unilateral pyelonephritis by removal of the affected kidney. A further success in relieving vascular hypertension by removal of a single chronically infected kidney has recently been reported by McIntyre (1939).

It may be thought that the removal of a focus of infection was in these cases responsible for a lowering of the blood pressure; but in view of the gross narrowing of the renal blood vessels which occurs in some cases of chronic pyelonephritis, and the success of Leadbetter and Burkland (1938) (to which reference is made below) in producing resolution of hypertension by removal of a single kidney in which only a vascular lesion was present, these isolated observations raise the suspicion that possibly obstruction to the blood flow may be the primary cause of the heightened blood pressure in these instances.

Moritz and Oldt (1937), discussing the pathological anatomy of arteriolar sclerosis in cases of hypertensive vascular disease, mention three elderly patients in whom chronic hypertension was not accompanied by arteriolar lesions but by sclerotic changes in the renal arteries which, the authors state, are "consistent with, although not definitely confirmatory of, reduced blood flow through the kidneys".

Boyd and Lewis record that the removal from a man, aged thirty-one years, of a kidney which was the site of an unsuspected infarct and in the blood vessels of which there were generalized degenerative changes together with narrowing of the vascular lumina, was followed by a return to normal of the blood pressure which previously had been raised. An X-ray examination revealed a heart of normal size, so in all probability the vascular hypertension was not of long duration and may have been the result of a reflex vasoconstriction arising in some way as a result of the infarction, rather than the outcome of an ischæmia due to the narrowing of the renal blood vessels, as the authors are inclined to believe.

An especially suggestive case was reported by Leadbetter and Burkland (1938) from the Johns Hopkins Hospital. A negro boy, aged five and a half years, had at the age of six months been found to possess an enlarged heart, and when aged three years to be the victim of vascular hypertension. Retrograde pyelography at the latter time revealed a misplaced right kidney overlying the promontory of the sacrum. No relationship between the ectopic kidney and the hypertension was suspected at this juncture. Two years later such a possibility was suggested and nephrectomy was performed. Following the operation the blood pressure fell to almost normal limits and remained low. The kidney was normal, as judged by microscopic examination, but the lumen of the main renal artery was in one place almost completely filled by a mass of involuntary muscle. Whether this sprang from the intima or media does not appear to have been determined; but whatever its origin, the illustrations shown by Leadbeater and Burkland indicate that it was without doubt adequate to produce a considerable degree of ischæmia in the ectopic kidney.

Leiter (1938) has published an account of three cases, one associated with obstruction of numerous arcuate arteries and interlobular arterioles, one with arteriosclerotic occlusion of the main renal arteries and one with fibrotic lesions of the kidneys and pronounced intimal thickening of the renal arteries and arterioles which he attributes to a congenital atrophy. In each instance vascular hypertension was present, and Leiter considers that the mechanism of its production was probably similar to that responsible for the high blood pressure in Goldblatt's laboratory dogs.

The importance of maldevelopment of the kidneys in association with hypertension in young persons has been discussed by Ask-Upmark (1929). While he records the advanced and extensive sclerotic changes in the small renal vessels in six persons between the ages of twelve and seventeen years in whom a high grade of vascular hypertension was present, he denies the possibility of a primary developmental defect of the blood vessels; but he considers that some abnormality of structure, which is often limited in extent, constitutes a *locus minoris resistentiæ* for the action of a toxic factor which produces as secondary effects the increase in blood pressure and the vascular sclerosis.

In order to accumulate data which may help to elucidate the suspected role played by obstruction to the renal blood flow in the genesis of increased systemic blood pressure, it may not be inopportune to consider, especially from the aspect of the pathological anatomy of the kidneys, cases of hypertensive vascular disease which are not associated with glomerulonephritis and which do not fall into the category of essential hypertension. It is chiefly with the hope of arousing interest in these less common but yet far from infrequent types of renal lesion associated with arterial hypertension that there are here recorded five cases which came to the notice of the writer while he was cataloguing the specimens illustrative of renal disease in the museum of pathology at the University of Sydney. In addition, there is described a case of renal dwarfism in which, despite a most extreme lack of renal secreting tissue, no hypertension had developed. This last case serves rather as a contrast to those preceding it, and illustrates that mere lack of renal parenchymal tissue does not of itself produce an increase in arterial blood pressure.

The clinical data are for the most part brief. This is partly due to the lack of complete investigation, but in part to the realization that the theme of this communication is of a pathological rather than of a clinical nature.

Reports of Cases.

CASE I.—*Senile Nephrosclerosis.* The patient was a woman, aged sixty-five years, who for six months had had a gradually extending œdema and became breathless even at rest. Her systolic blood pressure was 200 millimetres of mercury and her diastolic blood pressure 110 millimetres. During the three days preceding death the urinary output diminished till complete anuria developed on the last day. On her admission to hospital, three days before she died, the blood urea content was 207 milligrammes *per centum* and the blood creatinine content three milligrammes *per centum*.

At autopsy, ascites, hydrothorax and hydropericardium were found. The heart weighed 560 grammes and the left ventricle was hypertrophied. No gross valvular lesions were present. The right and left kidneys weighed 30 and 40 grammes respectively and their surfaces were irregularly scarred and granular. The cortical and medullary portions of the parenchyma were much shrunken and distorted, and several cysts of moderate size were exposed on the cut

surface. A most striking feature was the great thickening of the walls of the larger blood vessels, the cut ends of which projected above the surrounding tissue to an unusual degree. The gross appearances of the kidneys are shown in Figure I.

Microscopic examination revealed a most extreme grade of ischæmic fibrosis. The few surviving glomeruli appeared reasonably healthy. Thickening and fibrosis of the arterial walls were pronounced, and in places the lumina of the larger arteries had been occluded by organized thrombi, in which patchy calcification had developed. The thrombosis seemed to have occurred in atheromatous areas (Figure II). Some of the thrombosed vessels had been recanalized. Sclerosis of the smallest arterioles was not a conspicuous feature and, when it was present, was of a fibrotic rather than of a hyaline type.

The course of the disease in this case was suggestive of essential hypertension with terminal cardiac failure. With congestive failure of some six months' duration, the increased blood urea and blood creatinine contents were to be expected, even in the absence of a gross destruction of the secreting elements of the kidneys. Although information is lacking as to the health of the patient until six months before her death, the considerable enlargement of the heart in the absence of any valvular lesion suggests that the hypertension was of at least moderate duration.

Such small kidneys are, however, not usually found in essential hypertension, even when death is of uræmic origin. Moreover, the sclerotic lesions in the afferent arterioles are not of the hyaline type found in this disease. The preponderant vascular changes seem to be in the larger blood vessels, particularly in the primary and secondary branches of the renal arteries. In these vessels, atheromatous patches have led to thrombosis, and even in the vessels in which the thrombi have been recanalized, the lumina are so reduced that the ischæmia of the renal parenchyma must have been extreme. Such vascular lesions are of the variety commonly met with in nephrosclerosis of the senile type, with which increased blood pressure is not usually associated. Why in isolated cases such as this and in those reported by Moritz and Oldt (1937) obstruction in the larger branches of the renal artery should in the absence of pronounced arteriolar sclerosis be associated with increased blood pressure, it is difficult to say. Perhaps the resulting renal ischæmia more readily produces general vasoconstriction in some individuals than it does in the majority, so that these cases may really be equivalent to Goldblatt's (1938) examples of experimental hypertension. It is not possible, however, to deny that the hypertension may be of extrarenal origin; in this event the lack of arteriolar sclerosis may be due to the obstruction in the larger renal vessels shielding the smaller branches from the deleterious effects of a persistent hypertension. That such sclerotic and even necrotic lesions may be caused by high blood pressure, seems to have been proved by the observations of Goldblatt that in laboratory animals in which hypertension was prolonged the degenerative lesions in the small vessels of many organs resembled those found in human hypertensive disease of the malignant type, while in the kidneys, whose vessels were protected by the clamps applied to the renal arteries, such lesions were absent.

CASE II.—*Polyarteritis Nodosa.* A man, aged twenty-four years, developed severe aching pain in the shoulders and dimness of vision ten days before his admission to hospital. Physical examination revealed a systolic blood pressure of 240 millimetres of mercury, a diastolic pressure of 185 millimetres, and scattered hæmorrhages and patches of exudate in the ocular fundi. During the fourteen days between his admission to hospital and death the maximum blood urea nitrogen content was 60 milligrammes *per centum* and the blood creatinine content 1·5 milligrammes *per centum*.

At autopsy petechial hæmorrhages were found in the skin of various parts of the body. Along the course of the right coronary vessels were numerous small hard nodules raised above the surface of the heart. These proved on section to be aneurysms of the coronary artery (Figure III). The heart weighed 310 grammes, and there was no myocardial hypertrophy.

The general features of the kidneys are shown in Figure IV. They were of about normal size. The subcapsular surfaces were smooth, but coarsely mottled, with pallid and engorged areas and flecked with small hæmorrhagic spots which projected slightly above the surrounding tissue. The cut surface showed a similar mottling with scattered minute hæmorrhages. There was some loss of definition between the cortex and the medulla. Partially surrounding the left kidney, and lying between the capsule and the subcapsular surface was a large effusion of blood, which had caused some deformation of this kidney. The bleeding had apparently originated within the renal parenchyma, for towards the upper pole of the left kidney a channel filled with blood penetrated the substance of this organ and seemed to be continuous with the large effusion of blood already described.

On microscopic examination of the kidneys minute infarcts and numerous small hæmorrhages, chiefly intratubular, were found. Most of the glomerular tufts were relatively avascular, although there was a patchy congestion of the intertubular capillaries. A few tufts showed fairly advanced hyalinization. While no pronounced abnormality was found in the majority of the afferent arterioles, an occasional vessel of this group had necrotic walls. The arterioles of larger size and the small arteries had thickened intimal coats with minute lumina, and in some the muscular coat was largely replaced by a zone of fibrosis. Here and there necrosis of the wall of an interlobular arteriole could be observed, and in some instances the necrotic area was infiltrated with red blood cells, some of which had escaped into the surrounding interstitial tissue (Figure V). The lumen of one of the smaller branches of the renal artery was almost completely occluded by an extraordinary proliferation of fibrocelluar tissue in the intima, and its muscular wall almost completely replaced by fibrous tissue. At one point on the circumference of this vessel there was a distortion of structure, suggesting that aneurysmal dilatation had occurred before the proliferation of the intima had so much narrowed the lumen of the vessel (Figure VI). In places there was an overgrowth of fibrocellular connective tissue between the tubules in the cortex.

Both the general macroscopic appearances of, and histological changes in, the kidneys are similar to those seen in malignant nephrosclerosis. It must be pointed out that this term is used in a restricted sense, and not with the same irresponsible abandon which has characterized pathological and clinical literature within recent years. The concept of malignant nephrosclerosis and malignant hypertension would appear to include many distinct diseases which, resembling one another in age incidence, rapidity of progress, coexistence of increased blood pressure and development of renal failure, have too often been regarded as a clinical entity. A not inconsiderable proportion of those who suffer from "benign" hypertension die from renal failure if the destruction of renal tissue due to long-continued ischæmia is sufficiently great. Various observers place this proportion between 4·5% and 7%. It is customary in these instances to speak of the hypertensive disease as having passed into the malignant phase. In such cases the vascular lesions in the kidney are essentially the same as those found in the benign type, except that they are further advanced and more widespread. The most pronounced change in the vessels is a hyaline degeneration of the walls of the smallest arterioles, particularly of the afferent glomerular arterioles, with partial occlusion of their lumina. Even when the sclerotic changes in the larger vessels are pronounced, it is rarely difficult to appreciate the prominence of the hyalinization of the walls and the extreme narrowing of the channels of the arterioles of smallest size. It would seem that no essential modification of the pathological process found in benign hypertension had occurred, but that death had resulted from renal failure merely because the myocardium and the cerebral blood vessels had maintained their integrity relatively longer than in those patients who died from cardiac failure or following a cerebral catastrophe.

Keith, Wagener and Kernohan (1928) accept as "malignant hypertension" progressive hypertensive vascular disease associated with a generalized arteriolar hypertrophy in many organs and tissues, and with lesions of a sclerotic and degenerative nature in the retinal vessels which they regard as characteristic and distinguishable from the retinal lesions in glomerulonephritis. They group these cases together irrespective of the age incidence or of the mode of death, whether renal, cardiac or cerebral in type. Of the 81 cases

described by these authors, in only seven was an autopsy performed, so the possibility that the series includes diverse types of disease cannot be denied.

Ellis (1938), while recognizing the widespread occurrence of arteriolar necrosis in all organs in the condition that he refers to as malignant hypertension, holds that although death is due to renal failure, the lesions in the kidney develop late and may be slight in degree, and that in general examination of the kidneys at post-mortem examination reveals only slight or moderate contraction. The statement that necrosis of the blood vessels occurs in the kidney is difficult to reconcile with the comment that the renal lesion may be classified according to the system of Russell (1929) as chronic *nephritis repens*, Type IV. In the absence of detailed information one cannot be convinced that the 40 cases referred to by Ellis actually represent a disease entity and not a congeries of diseases associated with vascular hypertension of such a magnitude that secondary necroses have been produced in the blood vessels of numerous organs.

There occur, however, in individuals below the age at which essential hypertension usually develops, cases of hypertensive vascular disease which progress rapidly to a fatal outcome with failure of kidney function, and in which the characteristic lesion is necrosis of portion of the renal vascular tree, either of the capillaries of the glomerular tufts or of the afferent or interlobular arterioles. Resulting from these necrotic lesions in the walls of the blood vessels, there occur small hæmorrhages either into the lumina of the nephrons or into the interstitial tissue and also infarcts of microscopic size. Associated with these necrotic lesions in the vessels there is a widespread fibrocellular proliferation in the intima of the interlobular arterioles. If proliferative reactions appear in the glomeruli which at first sight suggest a glomerulonephritis, it will usually be found that they are associated with and may be regarded as a result of necroses in the glomerular capillary tuft. The vascular congestion around the infarcted areas and the infarcts themselves produce coarse reddish and yellow mottling of the kidney tissue, against which stand out in prominent relief the minute hæmorrhages springing from ruptured arterioles and capillaries. It was to this type of case that Fahr (1919) applied the epithet malignant. He insisted that these cases were not merely the end-result of a preceding benign hypertension, but constituted a disease *sui generis*, in the ætiology of which some toxic factor such as lead or lues played an essential part. While the nature and even the existence of the toxic element is now a matter of doubt, this general concept of a nosological entity is accepted by the writer, and it is this type to which in this paper the term malignant hypertension is applied.

It will be appreciated that the renal lesions described in the case at present under discussion correspond reasonably well with those accepted by Fahr (1919) as examples of malignant nephrosclerosis. In this instance, however, degenerative vascular lesions occurring in the coronary vessels have led to the formation of aneurysms, and it was no doubt from a similar aneurysm or area of necrosis in the wall of a small branch of the renal artery that the large subcapsular perirenal hæmorrhage developed. Though none of the histological preparations examined show the perivascular inflammatory areas often seen in *polyarteritis* (*periarteritis*) *nodosa*, there can be little doubt that a case of this disease is under discussion, for the vascular lesions are in general similar to those described by Arkin (1930), and in addition to the undeniable presence of aneurysms on the coronary arteries, one small vessel within the kidney (Figure VI) is the site of an abortive aneurysmal dilatation.

The acute onset with muscular pain is similar to that observed by Kernohan and Woltman (1938) and attributed by them to involvement in the pathological process of the small arteries or arterioles supplying the trunks of the peripheral nerves. These authors point out that the primary vascular lesion in Kussmaul's disease is a necrosis of the intima and media and that the periarteritic lesions are secondary to the degeneration in the vessel walls. If this is so, then the resemblance of *polyarteritis nodosa* to malignant hypertension is readily appreciated. In fact, it may be that some cases considered to be malignant hypertension are essentially cases of *polyarteritis nodosa* in which the lesions in the organs other than the kidneys are inconspicuous or lacking and in which rapid death from renal failure does not allow time for the development of aneurysms. It must, however, be recognized that arteriolar necrosis, whatever its ætiology, would produce similar lesions in the renal parenchyma, and the assumption of any relationship between these diseases must be purely speculative until the ætiology of each has been established.

Singer (1927) has reported two cases of *polyarteritis nodosa* in which the blood pressure reached high levels. Of 17 patients with this disease reported by Spiegel (1936), eight had hypertension; in one the blood pressure was normal, but in the remaining eight there was no record of the pressure. As the records of the autopsy findings in Spiegel's cases are noteworthy for their brevity, it would not be possible to correlate the incidence of renal vascular lesions with the presence of hypertension, even had the blood pressure been recorded in each instance. In only one of Singer's cases is the minute anatomy of the kidney described in any detail, and in this instance the occlusion of vessels either by thickening of their walls or by recanalized thrombi was a prominent feature.

Commenting upon these records, Fishberg (1939) states that "hypertension does not occur in *periarteritis nodosa* without extensive implication of the renal arteries". While this may be a statement of fact, it is based on no secure foundation, for little serious attempt has been made to collect data which bear on this question. In the case here reported, the obstructive vascular lesions were prominent and a considerable grade of hypertension had developed; but more information is needed before it will be possible to regard the hypertension occurring in the course of *polyarteritis nodosa* as the result of renal ischæmia due to the change in the arterial and arteriolar walls.

CASE III.—*Unilateral Renal Atrophy.* The patient was a woman, aged thirty-three years, who six years previously had had hæmaturia. Three years later a convulsive seizure had occurred during the birth of a child. For some years she had suffered from severe headache and vomiting, and her eyesight had been poor. Prior to her admission to hospital she had a convulsive seizure and became unconscious for two days. Her systolic blood pressure was 260 millimetres of mercury and her diastolic pressure 185 millimetres. Examination of the retinæ revealed abuminuric retinitis. On her admission to hospital the blood urea nitrogen content was 44 milligrammes *per centum*, but it rose to 142 milligrammes *per centum* ten days later.

At autopsy the heart weighed 270 grammes, the right kidney 240 grammes, and the left kidney 30 grammes. The right kidney (Figure VII) was enlarged, with a pale, slightly granular surface on which a few petechial hæmorrhages were to be seen. On the cut surface the cortex was pale, with distortion of the vascular pattern, and its average depth was somewhat increased. The bases of the pyramids were a little engorged, but the rest of the medulla was pale. The arcuate vessels were prominent, but their walls were not unduly thickened. The left kidney was very small, the renal parenchyma forming a narrow band only about one centimetre deep. A considerable proportion of the bulk of this small kidney seemed to be made up of branches of the renal artery with thickened fibrous walls.

Microscopic examination showed that the tissue of the small kidney was composed of densely packed atrophic and fibrosed glomeruli, sclerosed arterioles and interstitial fibrous tissue containing a few remnants of tubules. Vessels of the size usually assumed by those of arcuate type had undergone much intimal thickening, while the larger branches of the renal artery showed relatively little sclerotic change. One small artery had been recanalized and a new muscular coat had formed within the original *elastica interna*. The lumen of the vessel had also been almost obliterated by intimal proliferation. The extra-pelvic connective tissue was rather œdematous and contained only a few remnants of collecting tubules and dilated capillaries. A few isolated groups of lymphocytes could be seen in this tissue, but there was no convincing evidence of any active or past inflammatory reaction.

In the larger kidney many glomerular tufts were less vascular than normal, and an occasional adhesion could be seen between the tufts and the capsule. There was a

patchy fibrosis, the distribution of which suggested early hypertensive nephrosclerosis. A few afferent arterioles had thickened and hyaline walls. The interlobular and arcuate vessels had thickened intimal and somewhat fibrous middle coats. A number of arterioles were necrotic (Figure VIII), and this necrotizing change had involved the capillary tufts in some glomeruli. Hæmorrhage had occurred into the lumina of some nephrons.

There seems to be no evidence in this case which would justify any conclusion as to the cause of the atrophy or hypoplasia of the left kidney. The microscopic structure of this organ does not suggest that an infective process had been operating at an earlier period, but it would not be rational to exclude this possibility. A unilateral pyelonephritis may have produced such a scarred and shrunken kidney, and then in the course of time the infection may have died out, leaving no evidence of its previous existence. It would seem more likely, however, that the small size of the kidney is due to a hypoplasia similar in character to that occurring in Case V. The presence of considerable numbers of fibrosed glomeruli would suggest that the renal parenchyma had developed reasonably well, only to undergo a subsequent atrophy caused possibly by a local abiotrophy of the renal blood vessels.

The hæmaturia which occurred six years before death may have been due to an inflammatory lesion, but may equally well have resulted from rupture of blood vessels in the renal mucosa during the degeneration of the vascular system, of which ample evidence is to be seen in the tissue obtained after death.

While it is impossible to explain the reason for the atrophic state of the left kidney, the association of such unilateral renal atrophy with vascular hypertension and extreme grades of arteriolar sclerosis in the opposite kidney is of sufficiently frequent occurrence to arouse the suspicion that such a degenerative or hypoplastic lesion in one kidney may, by allowing the local formation of vasopressor substances, cause an increase in the systemic blood pressure, which in its early stages could be cured if the atrophic kidney were removed. Sometimes the unaffected kidney is the site of a hyaline arteriolar sclerosis similar to that found in benign hypertension; but in other instances frank necrosis of the small vessels is present, and the changes are similar to those seen in malignant nephrosclerosis.

CASE IV.—The patient was a man, aged thirty-eight years. In 1934 he had suffered from pyelitis due to *Bacillus coli communis* infection associated with frequent febrile attacks, during which his temperature rose to 105° F. A culture of the urine from the right kidney yielded *Bacillus coli communis*, while the urine from the left kidney was sterile. In a MacLean's test the maximum urea concentration was 2·6%. While the patient was receiving treatment for a hæmatemesis in 1936 his systolic blood pressure was found to be 130 millimetres of mercury and his diastolic pressure 90 millimetres. Excretion pyelography revealed no abnormality, but a urine culture yielded *Bacillus coli communis*.

In August, 1938, he came to hospital complaining of headache of eight months' duration and of poor vision, which he had first noticed six months previously. The systolic blood pressure was 280 millimetres of mercury and the diastolic pressure 160 millimetres. The blood urea content was 38 milligrammes *per centum* and the blood creatinine content was less than one milligramme *per centum*. The specific gravity of the urine varied from 1,008 to 1,020.

In February, 1939, he was once more in hospital. Since the previous admission three convulsive seizures had occurred, each preceded by a characteristic aura. On one occasion he had lost his sight for several minutes. Dyspnœa, palpitation and slight swelling of the feet had been present. The systolic blood pressure was 220 millimetres of mercury and the diastolic pressure 140 millimetres. The blood urea content was 43 milligrammes *per centum*. The urea concentration test revealed a maximum of 1·5%, and only 31% of phenolsulphonephthalein was excreted in two hours.

In July, 1939, his systolic pressure was 280 millimetres of mercury and his diastolic pressure 160 millimetres. The urinary specific gravity is recorded as 1,021 (single observation). The blood urea content varied between 210 milligrammes *per centum* and 265 milligrammes *per centum*, and the maximum blood creatinine content was 5·4 milligrammes *per centum*.

At autopsy the heart weighed 660 grammes and the left ventricle was much hypertrophied. The capsules of the kidneys were adherent to the perirenal tissues, so that removal of these organs was difficult. The left kidney (Figure IX) was deformed by several broad deep grooves and some less well-defined areas of depression between which the renal tissue projected in the form of large sessile nodules. The lower third of this kidney was free from such coarse scars, but was irregularly congested on the free surface. Here, too, some small hæmorrhagic spots were present. On the cut surface it could be seen that the depressed areas corresponded with zones in which the depth of the renal tissue was greatly diminished and the normal renal architecture grossly disorganized. The less affected portions of the renal parenchyma were irregularly congested, and the cortical markings were in some places distorted and in others obscured. The pelvic mucous membrane was rather opaque and for the most part slightly congested. In some calyces, however, an extreme degree of vascular congestion could be seen. Some of these calyces corresponded in situation with the depressed areas seen on the capsular surface. The appearances in the right kidney (Figure IX) were in general similar to those present in the left, but were of a much more advanced grade.

Microscopic examination of the less affected portions of the kidneys revealed an increase in the intertubular matrix, which was for the most part of a hyaline rather than of a fibrillary structure. In places the interstitial substance contained numbers of lymphocytes, a few plasma cells and an occasional polymorphonuclear leucocyte. Tubular atrophy of moderate grade had developed, and a few tubules contained numbers of degenerating leucocytes in a protein coagulum. Many glomeruli were apparently normal, but others showed partial hyalinization, adhesions to the capsule and diminished vascularity—in fact, changes similar to those seen in the glomeruli in glomerulonephritis (Figure X). A patchy capillary congestion was present. In the interlobular arterioles intimal proliferation was very prominent and in many of these vessels the lumen was almost completely occluded (Figure X). Similar but less striking changes were present in the arcuate vessels. In the deeply scarred areas the glomeruli were almost without exception completely fibrosed and densely packed together, forming a superficial zone in which relatively few tubular structures could be seen. More deeply situated was a region composed largely of acinar structures containing hyaline material embedded in a connective tissue stroma richly infiltrated with inflammatory cells (Figure XI). This constitutes the thyreoid-like zone described by Fahr (1938), Staemmler (1930) and others. Between this area and the cavity of the pelvis was a band of congested and in parts œdematous tissue containing partly destroyed and obstructed collecting tubules as well as numerous lymphocytes, plasma cells, histiocytes and polymorphonuclear leucocytes (Figure XII). The epithelium of the pelvic mucosa had in part been shed. In the blood vessels of all sizes intimal proliferation and fibrosis of the muscular coat were pronounced.

The chief interest in this case lies in the development in a relatively young person of arterial hypertension while renal function was still intact, consequent upon and presumably due to a persistent infection of the renal parenchyma. While it is manifestly impossible to form any opinion as to the condition of the renal blood vessels at the time of the development of hypertension, it is perhaps significant that even in these portions of the renal tissue, which at the time of death still contained many apparently intact secreting elements, the interlobular arterioles were extremely narrow as a result of a proliferation of fibrocellular tissue in the intima. Where the fibrosis of the kidneys is more advanced the vascular lesions are correspondingly more prominent. No gross lesions of the afferent glomerular vessels are to be found in these kidneys; but the intimal thickening of the larger blood vessels is so generalized that it is clear that the blood flow to the renal parenchyma must have been seriously impaired. As indicated in the introduction to this paper, it was possibly because Longcope was concerned chiefly with the afferent arterioles that he did not mention any correlation between vascular lesions and the incidence of hypertension in the chronic stage of pyelonephritis.

While it is not justifiable to seek in this single case any causative relationship between the vascular lesions in the kidneys and the hypertension, the extent of the narrowing of the arteries and arterioles is sufficiently striking to be of interest in view of Goldblatt's observations.

CASE V.—*Renal Hypoplasia.* The patient was a woman, aged thirty-seven years, who had suffered from breathlessness and epistaxis for five months, from precordial pain for one month and from frequent vomiting for six days. The systolic blood pressure was 200 millimetres of mercury and the diastolic pressure 120 millimetres. The blood urea content was 448 milligrammes *per centum* and the blood creatinine content 15·6 milligrammes *per centum*. The urine contained considerable quantities of albumin. Muscular twitchings developed before death.

At autopsy the heart weighed 400 grammes and the left ventricle was hypertrophied. Each kidney weighed 25 grammes. The subcapsular surfaces presented a moderately coarse granular appearance (Figure XIII). On the cut surface hardly any cortical tissue could be seen. The vascular pattern was grossly distorted, the boundary between cortex and medulla was obscured and even the pyramids were much deformed and shrunken. Several cysts were present in the parenchyma. There was an irregular distribution of blood vessels corresponding in size to the arcuate group. The walls of many of these vessels were thickened. The extra pelvic fat was relatively increased in amount.

On microscopic examination it was found that fibrous connective tissue in which numerous lymphocytes were present formed the bulk of the kidney. Those glomeruli which retained a relatively normal appearance had a pronounced pericapsular zone of fibrosis and were relatively few in number. Others showed hyalinization of the tufts and obliterative fibrosis in various stages. Atrophic and dilated tubules were to be seen, and in places the tubules were closely packed and filled with hyaline material producing an appearance suggesting thyreoid tissue (Figure XIV). Moderate fibrosis of the walls and narrowing of the lumina of blood vessels of all sizes had occurred. Calcified areas were numerous. The calcium salts seemed to have been deposited within the lumina of tubules; but in some instances basophilic material, which resembled calcium, occurred as aggregations of small globules in what appeared to be blood vessels. The significance of this latter change is obscure. The peripelvic connective tissue was fibrous, and though a few wandering cells were present, there was nothing to suggest an active or past inflammatory reaction (Figure XV).

The interest of this case lies in the problem of the nature of the pathological process from which the renal sclerosis has resulted. While the gross appearance of these organs is similar to that found in the chronic stage of a coarsely reticular focal glomerulonephritis, the reduction in size of the kidneys is much greater than is usually found in this condition. Histological evidence of glomerulitis is lacking, for such obliterative changes as can be found in the Malpighian bodies could be accounted for by ischæmia. It is, however, admitted that in the type of chronic glomerulonephritis described by Russell (1929) as chronic *nephritis repens*, Type IV, definite evidence of inflammatory lesions in the glomeruli may be absent. In the present instance, however, there are some unusual features. Glomeruli are present only in small groups beneath the capsule of the kidney, while over extensive areas densely packed atrophic tubules containing colloid-like material extend to the capsular surface without the intervention of any zone in which atrophied glomeruli can be distinguished. This structural feature corresponds with that described by Ask-Upmark (1929) and by Fahr (1938) as characteristic of developmentally hypoplastic kidneys. Extensive calcification is also rather more commonly met with in developmental abnormalities than in chronic glomerulonephritis.

The general tissue changes are somewhat similar to those observed in the atrophic areas of the kidneys in Case IV. In the case at present being considered there is, however, no microscopic evidence of any preexisting pyelonephritis. The fibrous zone immediately external to the pelvic epithelium contains an occasional lymphocyte; but the fibrosis may well be due to developmental defect and the wandering cells are insufficient in number to warrant a suspicion that they indicate an inflammatory reaction. Fahr (1938) has drawn attention to the similarity in microscopic structure of the kidney in chronic pyelonephritis and in hypogenetic kidneys, but considers that uniform scarring throughout the kidney and the absence of a well-defined superficial zone of fibrosed glomeruli are characteristic of the hypoplastic organ. The available evidence would suggest that in the present instance the renal lesions were developmental in nature. The presence of only a small number of obliterated glomeruli points to a primary hypoplasia of the secreting tissue; but presumably there has been an associated defect of the renal vascular tree, which has eventually led to such a degree of ischæmia that many of the surviving nephrons have atrophied. It is possible that the hypertension in this case, as in the previous cases in this series, may have been the result of such a diminished blood supply to the surviving renal tissue.

CASE VI.—*Renal Dwarfism.* The patient was a girl, aged sixteen years, whose physical development corresponded with that of a normal child of about nine years. Until three days before her death she was in her usual health. Then drowsiness and vomiting developed. Breathlessness was noticed two days later, and the drowsiness deepened into semiconsciousness. Physical examination revealed a brownish pigmentation of the skin over the whole body. The systolic blood pressure was 120 millimetres of mercury and the diastolic pressure 80 millimetres. Death occurred suddenly on the day of her admission to hospital.

At autopsy a distinct bilateral valgus deformity of the knee joints and a varus deformity of the ankles were found. The thoracic wall showed a "rickety rosary" at the costochondral junctions. In the thymic region was a fatty body with a few remnants of thymus tissue embedded in it. The heart was of about normal bulk in relation to bodily size, and weighed 160 grammes. Each kidney weighed 20 grammes and the capsules stripped easily. The surfaces of the kidneys were smooth, but on them congested vessels were prominent. On the cut face no differentiation into cortex and medulla was to be observed. The blood vessels were inconspicuous (Figure XVI).

Microscopic examination (Figure XVII) revealed a diffuse overgrowth of fairly mature fibrous connective tissue throughout the kidney. Most of the tubules were dilated and their epithelium was flattened. Evidence of tubular atrophy was almost completely lacking. Glomeruli were present in only small numbers, but almost no remnants of obliterated glomeruli could be found. Those present were hypertrophied and usually surrounded by a zone of concentric fibrosis. The capillary tufts were of normal vascularity and free from degenerative change. A few of the smallest arterioles had hypertrophied muscular walls; but for the most part the blood vessels were remarkably free from pathological changes. A few accumulations of lymphocytes were to be seen in the connective tissue, but there was no evidence of inflammation either present or preexisting. On the whole, the appearances suggested that the renal lesion was in the nature of a congenital hypoplasia of the parenchymal tissue.

This case of renal dwarfism is included as a contrast to those previously presented, as it is an example of failure of renal function without any concomitant vascular hypertension, apparently the result of an inadequate development of secreting tissue without any associated degeneration in vascular structures within the kidneys. Such cases indicate clearly that when increased blood pressure accompanies sclerotic lesions in the kidney, it cannot be attributed to the destruction of secreting elements. If, however, it is the ischæmia produced by sclerotic processes in the blood vessels of the kidney which causes indirectly the elevation of blood pressure, then it can be understood why in this case, in which such sclerotic vascular lesions were absent, no vascular hypertension was present. That the normal pressures observed were not merely due to a terminal fall from higher values is indicated by the small size of the heart.

Discussion.

As indicated previously, the object of this communication is to draw attention to certain varieties of renal lesion which are at times unexpectedly found at autopsies upon individuals who have suffered from vascular hypertension. There is perhaps an undue readiness to diagnose as chronic glomerulonephritis or essential hypertension many obscure disorders associated with an elevated systemic blood pressure without due regard to the diversity of factors to which such hypertension may be due. Unless every effort is made to segregate into separate classes, by the employment of all available aids to diagnosis and by correlating whenever possible the clinical findings with what is revealed at autopsy, those cases presenting unusual features, we shall probably lose numerous opportunities

of throwing a gleam of light on the many obscure aspects of hypertensive vascular disease. In this survey we are concerned with this question only in so far as it concerns the kidneys. A particular aspect of this problem which may well be mentioned is the clarification of the concept of malignant hypertension. Enough has already been said to indicate the confusion to which the more or less indiscriminate and illogical use of this term has led. There can be no justification for the characterization as "malignant hypertension" of all cases of hypertensive vascular disease, unassociated with an obvious cause such as an adrenal tumour, occurring in young or relatively young people and making rapid clinical progress. Some of these cases may be the result of developmental abnormalities in one or both kidneys or in the renal blood vessels; others may be due to chronic infective renal lesions or to systematic vascular diseases akin to *polyarteritis nodosa*; others again are no doubt unrecognized cases of glomerulonephritis. Only by a systematic assembling and surveying of all data—clinical, physiological and anatomical—in cases in which the diagnosis of malignant hypertension is made, will it be possible to determine whether there exists a clearly defined entity to which the term may justifiably be applied. In Cases II, III, IV and V of the present series, "malignant hypertension" was considered as the most satisfactory clinical diagnosis; yet in no two of these instances were the renal lesions similar. Only by the most complete investigation of the functional and structural characteristics of the kidneys during life would it be possible in certain of these cases to determine their true nature.

It must be admitted that with our present lack of suitable technical methods for investigating in detail the functional disturbances of the kidneys, such investigations are severely handicapped. There are, however, certain simple procedures, the more general application of which might lead to the recognition of some conditions which at present escape detection. No case should be diagnosed as chronic glomerulonephritis unless the possibility of an infection of one or both kidneys is first of all excluded. It is customary to inquire about the occurrence of scarlet fever or rheumatic fever when golmerulonephritis is suspected; but how often in such a circumstance is any inquiry made about the presence at some earlier time of acute cystitis or pyelitis? Even if no such information can be elicited, a bacteriological examination of the urine should be made to exclude the likelihood of chronic pyelonephritis. If the presence of an infective condition is established, there is little doubt that an attempt should be made to discover whether or not the infection is unilateral. The successes which have so far followed the removal of a single infected kidney call for a more general recognition of this procedure.

In what are usually considered medical as distinct from surgical diseases of the kidneys, the use of excretion or retrograde pyelography is rare. Were these methods of investigation more widely employed, it is probable that, not infrequently, unilateral renal lesions would be found in association with vascular hypertension. Surgical removal of the diseased or malformed kidneys might in these cases save the patient from a premature death. Since it has been shown by Homer Smith (1939) that "Diodrast", an organic iodine compound used for excretion pyelography, is quantitatively removed from the blood during a single passage through the kidneys, and since the concentration of this material can be estimated in the blood and urine, a determination of the "'Diodrast' clearance" in a manner similar to the estimation of the "urea clearance" enables a measurement of the renal blood flow to be made. At present this procedure is limited in its application because of the technical difficulties involved in the "Diodrast" estimations; but if these estimations can be simplified, then a widespread employment of this means of measuring the rate of blood flow through the kidneys may elucidate many problems of renal pathology.

In the five cases of hypertension described in this paper, structural changes in the blood vessels competent to produce ischæmia of the kidneys were present. Although in the sixth case the reduction in renal secreting tissue was extreme, no obstruction to the flow of blood could be found, and in this case there was no hypertension. This in itself proves nothing, but is perhaps an encouragement to seek a correlation between the hindrance to the flow of blood in the renal vascular tree and the occurrence of vascular hypertension. Even if such a correlation can be established on a morphological basis, it merely strengthens the belief, but does not prove the thesis, that in the human subject, as in the laboratory animal, renal ischæmia can produce an elevation of the systemic blood pressure. It may reasonably be argued that the sclerotic changes in the renal vessels are the result and not the cause of the hypertension. There seems but one way to investigate this question in man: that is, by measurement of the renal blood flow at intervals in cases of progressive renal disease and correlation of any restriction of the flow with the development of hypertension.

It is not reasonable to assume that because renal ischæmia is a cause of vascular hypertension it could be demonstrated in more than a fraction of hypertensive cases. In our present state of ignorance it is not possible to deny that there may be almost as many factors competent to produce an elevation of blood pressure as to cause an elevation of body temperature. Goldblatt's work, however, shows beyond cavil that hypertension can be caused by restriction of the blood supply to the kidneys. It is only by the cooperation of the clinician, the pathologist and the biochemist that there can be a reasonable hope of determining whether in human patients a similar mechanism can be demonstrated, and of recognizing those cases in which treatment can be applied to shatter this deleterious sequence of cause and effect and so spare the entire vascular system from the damaging effects of prolonged hypertension.

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A NOTE ON LIPOID-CELL PNEUMONIA.

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THE condition known as lipoid-cell pneumonia is described as resulting from the involuntary inspiration of oil given as medicine or in food. Cod-liver oil and paraffin are indicated as the chief offenders, but this is perhaps only because they are the most commonly used. Experimentally it has been produced also in different degrees by olive, linseed, castor and poppy-seed oils, by butter fats and esters, and by lecithin; cholesterol has little action under the conditions of experiment (Paterson, 1938).[2] In general it is stated that vegetable oils are the least, and animal oils the most intensive in their action (McCallum, 1939).[1]

The nature of the lesions is described by the above-mentioned and other authors, so that here I shall give in brief only the results of observations made in three cases recently met with in this hospital. The inspiration of the oil seems at first simply to flood the alveoli, to which it is conducted by a bronchus. This leads to oil-soaked patches of lung which in autopsy specimens (that is, when "set") are greyish white, consolidated masses having the appearance of lard. Examination of microscopic sections reveals some thickening of the alveolar walls, and the contained air spaces are more or less filled with oil globules of various sizes and shed alveoli cells. The latter, acting as macrophages, break up the droplets and stuff themselves full of fat granules (lipoid cells). Presumably they enter the blood and carry the fat to a place of destruction; in the cases under discussion no oil was found in the bronchial lymph nodes. In places the alveolar epithelium had fused into giant cells for "massed attack" as in other reticuloses. Here and there was found some superadded pneumonia of ordinary type, showing exudation of leucocytes and serum with œdema; the result, one supposes, of secondary infection. No doubt some of the inspired oil is returned by the cilia and bronchial muscular action; but on entering the alveoli it is beyond the reach of these defences, and its removal then becomes the task of the macrophages and their associated biochemical processes.

The reports hitherto available to us are concerned mostly with the natural and experimental pathology of the malady, and not much clinical information has been gathered. In our own three cases the children were all sickly; cough and chest signs were always present; dulness, bronchial breathing and râles were noted, but these were not distinctive, and were attributed to the bronchopneumonic conditions from which our patients so commonly suffer. This was also the diagnosis from the X-ray examination. It is to be presumed that the entrance of the oil into the larynx and trachea would stir the usual physiological attempts at removal, and doubtless some oil was regurgitated. It is stated that oil drops may be found in the sputum when available—that is, in the sputum of adults and older children. The younger ones do not often oblige with this material, and there was no opportunity of making such examinations. In a subsequent suspected case oil droplets were found in fluid obtained by lung puncture. However this may be, although cough was present, there was nothing special about it. A "whoop" was mentioned in one case, but not very emphatically, and there was no real question of pertussis. There were no signs directing attention to the "foreign body"—no "gagging" or other indication of anything "going down the wrong way". The process was clinically silent.

This inspiration of oil is said to be specially likely when defects are present which interfere with proper deglutition. Vomiting, regurgitation, convulsions, paralysis, dulled reflexes, cleft palate *et cetera*, are mentioned in this connexion. In quite a considerable number of reported cases the lesion followed nasal instillation of oily medicaments. One of our patients who had vomited daily since birth and could not sit up was given nasal drops in paraffin. One other vomited frequently and had to be tube fed. The third had congenital diplegia. Both the last two patients had "emulsion" as part of their routine feeding. Reports do not make it clear that defects are essential; the sudden insuck which follows vomiting seems to have sufficed.

At the moment the lethality of the condition is not apparent, and presumably recovery occurs from minor degrees. In several instances small oily areas have been found unexpectedly in lungs during microscopic examination; seemingly these were early stages of the process. But obviously the lesion can progress to dangerous occlusion of the breathing space in the lung. So it would be prudent to have it in mind in all cases when patients are taking oil, to be on the watch for bronchopneumonic signs, and to accept them as a warning of possible impending lipoid-cell pneumonia.

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Reports of Cases.

A PROCTOLOGICAL PROBLEM: CARCINOMA OF THE RECTUM AND HYDATIDOSIS OF THE PELVIC BONES.

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DR. G. M. OXER, in the issue of THE MEDICAL JOURNAL OF AUSTRALIA of April 6, 1940, at page 483, reported the case of a patient, J.P., who was initially treated at the Royal Melbourne Hospital for a condition regarded as carcinoma of the rectum, and who died seventeen years later at the Austin Hospital with hydatidosis of the pelvic bones.

Dr. Oxer has obtained records from the Royal Melbourne Hospital which are obviously incomplete, with the result

that several inaccuracies are present in the history he records. He has concluded from the incomplete data at his disposal that the condition of *carcinoma recti* did not in fact ever exist, a finding which, while possibly correct, is not in accord with the weight of evidence available. In these circumstances the patient's history is rewritten in detail.

Summary.

This case of carcinoma of the rectum occurred in a young man, aged twenty-nine years. The diagnosis was made at sigmoidoscopic examination, and an operation for excision of the rectum was commenced and abandoned. The disease was found inoperable, not because of the extent of the primary growth, but because of the presence of a secondary mass adherent to the pelvic wall.

The patient was treated with X-ray therapy, following upon the performance of a permanent colostomy. The carcinoma resolved, leaving a fibrous stricture, and the patient remained well of his disease for sixteen years.

His death was due to pulmonary complications. Hydatid disease of the right *os innominatum* coexisted; symptoms appeared about five years previously. The radiological appearances of this disease were wrongly diagnosed as those of secondary carcinomatosis, and the true nature of the pathological process remained unsuspected during life. At post-mortem examination hydatid disease was found to in no other site; and the case would appear to be one of primary hydatid disease of bone. The post-mortem diagnosis, therefore, should read: "A case of carcinoma of the rectum, in which cure was effected and in which death was due to primary hydatid disease of the pelvic bones and pulmonary abscesses."

Details of the Clinical History.

The patient was first admitted to hospital on July 9, 1923. He complained of a frequent desire for defæcation, present during the previous twelve months. The fæces were usually dry and hard, and straining caused pain and irritation. Blood and mucus had occasionally been seen. He was often at stool four or five times in the night, and conditions had become much worse in the last three months. Micturition was normal. He had lost one stone in weight in the twelve months. He had previously lived in Egypt for a year, but had not contracted any bowel infection. The rest of his life had been spent in the Melbourne suburbs.

The provisional diagnosis made on his admission to hospital was "prolapsed rectum for investigation". Examination revealed a mentally retarded man, aged twenty-nine years. Abdominal examination revealed no liver enlargement; the descending colon was loaded with fæces and was somewhat tender. When a rectal examination was made, a hard fibrous ring was found surrounding the lumen of the bowel, about three inches up from the sphincter, and projecting mostly from the posterior wall. The mucous membrane below the stricture appeared polypoid. General examination otherwise revealed no abnormality.

On July 26, 1923, Mr. Harold Dew examined the rectum with a sigmoidoscope. A ring carcinoma was revealed about five inches up, surrounding the rectum and greatly encroaching on the gut. Attempts to obtain a piece of the lesion for biopsy examination were unsuccessful.

At operation on August 2, 1923, Sir Alan Newton made a mid-line incision and opened the abdomen. The patient was placed in the Trendelenburg position, and the rectal tumour was found to be a large fleshy growth involving the lumen, and with secondary sowing on the peritoneal serous coat. The involved bowel was considered to be easily removable, but a large hemispherical secondary growth was found occupying the *obturator foramen*. The abdomen was therefore closed after a first-stage colostomy had been performed by means of a muscle-splitting incision. Three days later the bowel was opened, and on August 15, 1923, the patient was transferred to the care of Dr. L. J. Clendinnen for deep X-ray therapy. Deep X-ray therapy was ordered on four occasions during August and September, 1923, and on two occasions during January and February, 1924.

A definite improvement became obvious after the initial therapy, and the appearance of the condition some months later was clinically consistent with a hard fibrous stricture.

In August, 1924, an examination with a proctoscope was made by Dr. J. G. Whitaker. A specimen from the edge of the stricture was forwarded to Dr. Mollison for histological examination. He reported that the section "shows an alveolar stroma with masses of cells, hardly recognizable as carcinoma, though it probably is one". At this time these appearances were interpreted as revealing damaged carcinoma cells "enbalmed" in fibrous tissue; and this was regarded as a very satisfactory result from the radiation therapy. It was thought, however, that this interference with the quiescent lesion might stir up further activity; and two further prophylactic treatments were administered before the end of the year. A further course of four treatments was given in July and August, 1925; and this concluded the radiation treatment to the primary lesion.

Generally the patient remained well; but his colostomy gave him considerable trouble, on account of prolapse of the mucosa through the opening, and he was subject to continual bouts of uncontrollable diarrhœa. He was admitted to the surgical ward on three occasions for operations for repair of the colostomy.

On June 27, 1927, Sir Alan Newton made a proctoscopic examination and found a fibrous stricture in the old site of involvement, and a specimen from this was sent for biopsy. This section showed no evidence of carcinoma; but some hyaline fibrosis consistent with post-radiational change was present. At this time nothing further was done.

The patient was admitted to hospital again on December 14, 1930, when an operation for repair of the prolapsed colostomy was carried out by Sir Alan Newton. General examination revealed no abnormality, and rectal investigation revealed a stricture with a lumen narrowed to half an inch in diameter. No mass was felt.

The patient was readmitted to hospital again three months later, as further prolapse had occurred, which he had been unable to reduce. A further plastic repair of the prolapse was undertaken and the patient was discharged from hospital a fortnight later.

During the next two years the patient complained of intermittent diarrhœa, with the passage of some blood-stained mucus from the rectum. Proctoscopic examination by me on May 30, 1932, revealed a few papillomatous outgrowths from the ampullary mucosa. The condition of the stricture remained the same and the papillomatosis was held to account for the hæmorrhage.

In October, 1927, the patient first complained of right-sided pelvic pain; but no abnormality was found in the pelvic bones on radiological examination.

In April, 1935, twelve years after the diagnosis and treatment of his carcinoma of the rectum, his pelvis was reexamined radiologically on account of pain in the right hip and buttock. These films showed that a condition of patchy rarefaction was present in the right ilium, which was regarded as secondary carcinomatous involvement (Figure I). The length of this latent period, the fact that the original obturator gland was attached to the opposite wall of the pelvis, and the rarity of secondary bony metastases from carcinoma of the rectum prompted an unsuccessful search for another primary neoplasm. A fractionated course of deep X-ray therapy was ordered by me to this area at this time and was repeated five months later. Little relief to the patient was evident and further radiographs showed progressive changes with involvement of the acetabulum, the ischium and the head and neck of the femur. He refused further therapy, as he could not lie with comfort on the treatment table.

His pain gradually became worse, and as his people could not manage him at home, he was admitted to the Austin Hospital early in 1938 with a diagnosis of advanced secondary malignant disease involving the right ilium and ischium. X-ray examination revealed no abnormality in the lungs, and the radiologist (Dr. Mackay) was close to the truth in reporting that the widespread destructive lesions in the bones comprising the hip joint suggested an inflammatory rather than a neoplastic process. After three months of treatment along general lines he was discharged from hospital, clinically improved.

Six weeks later he reported back at the Royal Melbourne Hospital, complaining of pain in the lower part of the right leg and much discharge *per rectum*. Wasting of the thigh and shortening of the leg were present. Some sixteen months later he was again admitted to the Austin Hospital, having received one course of deep X-ray therapy at the Royal Melbourne Hospital in the interim.

On his readmission to hospital on April 28, 1939, he had a fairly severe cough, which, he stated, had commenced two months before, when he had got wet and caught a chill. Examination disclosed no abnormality of heart or lungs, and the liver was impalpable. The right thigh was flexed and internally rotated, but not fixed, and some muscular wasting was present in that area. Rectal examination, as on recent previous occasions, could not be carried out on account of the severe pain produced by the attempt.

The radiologist's report on the pelvis at this time read as follows:

> Metastases in the right half of the pelvis with destruction of the acetabulum and inward dislocation of the femur. Metastases also in the upper end of the femur. Slow progression of metastases since previous report.

Secondary deposits in the lungs were suspected, as the cough failed to improve, and a skiagram revealed an area

of dulness toward the lower half of the right lung. These appearances were consistent with metastases and associated atelectasis and some inflammatory changes.

After some weeks the patient experienced a recurrence of the previously troublesome diarrhœa, which no symptomatic treatment completely controlled. His sputum was fairly profuse and of purulent appearance, but contained no tubercle bacilli. The Wassermann test produced no reaction. His temperature was always within normal limits and his pulse rate varied from 40 to 104 beats per minute, usually 55 to 70. Two weeks after the onset of the diarrhœa, on June 24, 1939, he died.

Post-Mortem Examination.

Autopsy was carried out on June 25, 1939, by Dr. G. M. Oxer. The posterior and basal parts of the left lung were very œdematous, the lung being otherwise healthy. The lower lobe of the right lung was adherent to the lateral thoracic wall and was riddled with abscesses, the largest being about three centimetres in diameter. No bronchial obstruction or gross evidence of malignant disease was found. The liver was soft and engorged, but there were no carcinomatous deposits here, nor in any other of the abdominal viscera or glands. Evidence of a recent generalized fibrinous peritonitis was present, but the intestines were otherwise normal. The rectal lumen was diminished in one place to about one centimetre in diameter, but otherwise the organ was normal except for a very minor degree of polyposis.

Incision over the right pelvic bones disclosed a mass of hydatid cysts, and further dissection showed that the whole right side of the pelvis and the right femur were riddled with hydatids, both inside and around all of the bones and within some of the muscles.

Comment on the Diagnosis of Carcinoma of the Rectum.

It cannot be dogmatically stated that a carcinoma did exist, as no section of the untreated lesion was ever obtained.

However, considerable weight must be attached to the clinical evidence of Mr. H. R. Dew, who on proctoscopic examination observed "a ring carcinoma about five inches up . . ." The patient then came under the care of Sir Alan Newton, who began an operation for radical removal of the rectum. He found a fleshy growth involving the lumen, with secondary sowing on the peritoneal serous coat and a large secondary deposit in the obturator fossa. Radical removal was impracticable and the operation was abandoned.

Deep X-ray therapy was administered and the growth regressed; but a stricture remained at the ring site, and from this some considerable time later a section was obtained. Dr. Mollison reported the presence of cells, probably carcinomatous, in a dense fibrous stoma. This report is chronologically misplaced in Dr. Oxer's history and is consistent with a successful radiotherapeutic clinical result. Unfortunately, the section has been lost and the block destroyed. Further deep X-ray therapy was given and the patient, if he was suffering from carcinoma, remained cured.

Papillomatosis of the rectum is known to have coexisted, and this condition may provide an ætiological basis for the neoplasm.

If this case was not one of carcinoma, what was the true diagnosis? How else can the primary fleshy mass with a stricture, the peritoneal sowing and the secondary deposit in a gland be explained?

The growth responded to radiation therapy. *Carcinoma recti* is not classed among the sensitive neoplasms, but many cures are recorded, and cure therefore is possible. Here the response to the small total dosage (by present-day standards) can be explained only by the massive unit doses and by abnormal radio-sensitivity of a carcinoma in a very young man.

But if the condition was not a neoplasm, why did radiation therapy achieve any result? A later section from the stricture site shows fibrosis only; but in this section hyalinization is present, which is consistent with scar tissue formed as a result of radiation treatment.

The patient's first complaint of pain in the right lower extremity was made in 1927, four years after his original operation, and radiological examination at the time revealed no abnormality. No further complaint of pain in this area was made till 1935, seven and a half years later, and at the time radiological evidence of bone destruction was demonstrated. Thus at laparotomy in 1923 the pelvic cavity was obviously free of any hydatid deposit and in 1927 the pelvic bones were free, and so the hydatid process probably commenced in the years immediately preceding 1935, at least ten years after the treatment of the carcinoma.

Note on the Deep X-Ray Therapy.

The initial course of four treatments with deep X rays was given in August, 1923, before 200-kilovolt apparatus had been installed in the hospital. Therapy at that time was administered by a converted Waite and Bartlett radiographic unit equipped with a Coolidge tube and working at 100 kilovolts and 10 milliamperes. No details of the doses given are available, but the filter used was five millimetres of aluminium. From 1924 onwards the treatment was carried out by a Waite and Bartlett 200-kilovolt mechanically rectified unit.

The treatment of this patient illustrates the Erlangen technique, in which massive single doses were given to two or three large fields, the skin dose being carried to the extreme limits of cutaneous tolerance. A prolonged period of rest followed for skin recovery before the administration of further doses of a smaller order.

The dosage administered to the supposed secondary malignant involvement of the right side of the pelvis followed the present-day divided dose method. Secondary carcinoma of bone usually responds promptly to a tumour dosage of the order of 1,000 to 1,500 r; this is shown by immediate relief of pain and regeneration of bone in the rarefied areas. This patient had neither alleviation of the pain nor arrest of the bony destruction as a result of the deep X-ray therapy; these facts are readily explained by the post-mortem examination.

This lack of response prompted the prescription of larger doses in subsequent courses of treatment, in an effort to arrest the progress of the disease. The radio-resistance of the supposed secondary involvement in comparison with the obvious radio-sensitivity of the primary lesion aroused comment at this time, especially as bone metastases are usually more sensitive than their parent neoplasm.

Acknowledgements.

I wish to express my thanks to Dr. A. M. Hutson, Medical Superintendent of the Austin Hospital, who has gone to much trouble in supplying me with detailed notes of the patient's history from the records of the Austin Hospital. Thanks are also due to Miss Waud, who has made the X-ray prints for me.

Reviews.

ANATOMY.

IN recent years several attempts have been made to present in a handy volume the fundamental facts or principles of anatomy. Each has been good so far as it goes, and probably embodies the facts presented by the author concerned in a course devised for a particular group of students. The present volume is a commendable attempt to present to an elementary class, whose members have had no previous biological training, the most fundamental facts of anatomy, and should serve as a helpful introduction to the subject.[1]

In all sections the subject matter is very simply presented, should be followed without difficulty, and for the most part is accurate; but without putting undue strain on the intelligence of an elementary class, certain sections could with profit have been more fully developed. This applies particularly to the section on the development and growth of bone, which could have been expanded in the interest of greater accuracy than that shown in Figure V (E), and to the section on the muscular system, where the treatment of the histological facts might perhaps have been a little fuller.

The section on the nervous system is a very succinct account of the main structural facts.

The illustrations, although extremely diagrammatic, are very useful in an introductory book of this sort, which is obviously prepared merely to present essentials to students who have access to the dissecting room and to works with accurate illustrations. Reference has already been made to Figure V (E), and a few other drawings could be improved upon or omitted—for example, Figure XXI (E), Figure XXV (B) and (C).

On the whole a useful body of readily assimilable facts is here presented in a book which is well printed on good paper and well bound.

[1] "The Principles of Anatomy: An Introduction to Human Biology", by A. A. Abbie, M.D., B.Sc., Ph.D.; 1940. Sydney: Angus and Robertson Limited. Medium 8vo, pp. 143, with illustrations. Price: 1/6s. 6d. net.

The Medical Journal of Australia

SATURDAY, DECEMBER 14, 1940.

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THE TREATMENT OF CEREBRO-SPINAL MENINGITIS.

IN his comprehensive paper on cerebro-spinal meningitis publisned in this journal at the end of last July, W. L. Calov referred briefly to methods of treatment. First of all he mentioned lumbar puncture as a therapeutic as well as a diagnostic measure, and then he discussed antiserum and antitoxin. He reminded readers that it was not until the great epidemics of 1915 and 1916 that the real value of antiserum was demonstrated. He explained that antiserum was given intravenously in the bacteriæmic stage and intrathecally or both intrathecally and intravenously in the stage of meningitis. He also laid emphasis on the fact that sulphanilamide drugs have revolutionized the treatment of cerebro-spinal meningitis, and quoted the findings of several authors in support of his remarks. A great deal has been written on the treatment of cerebro-spinal meningitis; clinicians searching for reliable information may easily be lost in the mass of material that they will find in the journals commonly available in this country. Perhaps one of the most important contributions of recent years is that of H. Stanley Banks, published in *The Lancet* of July 2, 1938. His paper was called "a preliminary survey based on 113 cases", and may be used at the present time as a basis for any study of the subject. The treatment of cerebro-spinal meningitis, which is not a simple matter, should be considered by Australian practitioners, and that they may have their interest stimulated their attention is directed to a discussion held last May at the Royal Society of Medicine.[1]

It will be appropriate at the outset to refer to a statement by the last speaker in the discussion, Mervyn Gordon, who said that there was at the present time too great a difference between the mortality rates of patients treated by experts and those treated by others. He entered a plea for the creation of special machinery to disseminate knowledge of the subject. This dissemination can be done by medical journals. Unfortunately the expertness desired by Gordon can be obtained only by those who have to treat numbers of affected persons. In other words, the occurrence of many sporadic cases or of an epidemic is necessary. At the same time a good deal can be done by what has been called armchair study. A student of the literature is far better equipped to treat a sporadic case that may fall to his lot than is the man who leaves his reading until he is faced with an obvious diagnosis, or has perhaps already made one or two diagnostic errors in the presence of an epidemic.

H. S. Banks, whose paper in *The Lancet* has already been mentioned, opened the discussion at the London meeting; his indeed was the chief contribution. He based his remarks on two series of cases totalling 320, and the first fact that should be noted is that the fatality rate for these cases was not more than 10% or 11%. This rate is in striking contrast to that of 22% for the whole of England and Wales. These figures would appear to justify Gordon's remarks on expert treatment; and we may therefore regard Banks's detailed instructions in the use of chemotherapy as worthy of careful attention. Before these details are given it is necessary to point out that Banks is becoming more and more inclined to rely on chemotherapy alone and to discard the use of serum. In his article of July, 1938, he stated that he regarded the intrathecal use of serum as obsolete; but he recommended the administration of one or two large doses by the intravenous or intraperitoneal route. He held that when serum was given in this way it penetrated rapidly to the cerebro-spinal fluid and was maintained there in small amounts for many days. In October, 1939, Banks wrote in *The Lancet* of the "auxiliary value" of serum, and now he states that "in spite of theoretical considerations and experimental evidence" he is "not at present convinced that serum administration is necessary or desirable, even as an adjuvant to chemotherapy". This statement is made after reference to two tables that deal with two groups of sporadic and one group of epidemic cases that occurred at different periods. Some of the observations on these cases are full of interest, but the tables will not stand up to a critical analysis—such observations as six cases, two deaths and a gross fatality rate of 33·3% should find no place in the writings of so obviously capable a man as H. S. Banks. Among the notable observations are the severity of the epidemic as compared with the sporadic cases, and the fact that no less than "20%" of the epidemic cases were not verified by bacteriological examination on account of the rapidity of sterilization of the cerebro-spinal fluid under chemotherapy in a number of cases in which patients had received treatment before they were admitted to hospital.

In his summary of treatment Banks gives useful directions. In the first place there are two drugs to be used, and each has its indications. Sulphapyridine given by mouth is "the first choice". If it causes vomiting twice, nausea, depression or hæmaturia, it should not be used further, nor should the sodium salt be injected. These symptoms are indications for a change to sulphanilamide. As sulphanilamide is less potent than sulphapyridine, the dosage must be adequate. Banks points out that there is some evidence that sulphathiazole is as potent against meningococci as sulphapyridine, but less

[1] *Proceedings of the Royal Society of Medicine*, July, 1940.

liable to cause vomiting; he thinks that it may become "the drug of choice". The dosage recommended is set out in a table. There are three periods of administration. The "initial period" varies from two days in mild cases to three days in severe infections. The daily dose ranges from three grammes for children less than a year old to nine grammes for those over fifteen years of age. The "middle period" consists of two days during which two-thirds of the dose is given; the "final period" is two days, and one-third of the dose is given. The duration of administration is thus six to seven days, and adults receive from 36 to 50 grammes. The dosage is that for sulphanilamide; it is stated to be somewhat high for sulphapyridine, but to be safe if it is not exceeded. In the average case the duration of treatment should not exceed six to seven days. If the infection is very acute Banks recommends for an adult two initial doses of two grammes of sodium solution of sulphapyridine given intravenously and diluted in three volumes of saline solution. Intramuscular injection of the sodium solution should be used only when there is no other alternative, on account of the necrosis of muscles that it causes. Spinal drainage is sometimes required to relieve pressure symptoms, and fluid must be given to prevent or relieve dehydration. Banks gives no directions regarding serum, but G. Emrys Harries, reporting on two hundred patients treated at the City Isolation Hospital, Cardiff, said that 30 to 60 cubic centimetres of meningococcal antitoxin were given intravenously in normal saline solution in severe cases. It is interesting to record that at the Queen's Memorial Infectious Diseases Hospital, Fairfield, Victoria, F. V. Scholes is obtaining very good results with the use of "M & B 693" together with drainage by lumbar puncture. Since the use of "M & B 693" was started at Fairfield, serum has not been used, except in one or two cases. Before "M & B 693" was introduced antimeningococcal antitoxic serum was used at this institution together with antibacterial serum and lumbar puncture; in a series of thirteen cases only one death occurred when this therapeutic combination was used.

The question as to whether serum should or should not be used cannot at present be decided—it would not be safe to make a dogmatic statement. There is no evidence that in every type of case the use of the drug alone is as satisfactory as the combined use of drug and serum therapy (together with lumbar puncture, of course, when necessary). On theoretical considerations it is reasonable to use antibacterial and antitoxic serum, and it is a fact that in animals the combination of drug and serum is better than the use of the drug alone. In a recent personal communication Scholes has stated that "M & B 693", together with lumbar drainage, is producing very good results in ordinary cases. His view is that if and when the virulence of the meningococcus becomes exalted to the degree that it attained in 1916-1918 as an accompaniment to starvation, dirt and destitution, and a preponderance of really malignant cases occurs, it will then be possible to estimate the true value of "M & B 693" and the advisability or otherwise of using serum in addition. He thinks that if fulminating cases occur and the patients are seen at a sufficiently early stage, clinicians will be glad to use antitoxic and antibacterial serum intravenously, and he himself proposes to do so. Australian practitioners will accept the views put forward by Scholes.

Current Comment.

GOVERNMENT MEDICAL SERVICE IN TASMANIAN COUNTRY CENTRES.

AT this time last year we drew attention to the annual report for the year 1938 of Dr. B. M. Carruthers, Director of Public Health of Tasmania, and in particular to his summary of the work carried out by the whole-time Government medical officers in country centres in different parts of the State. It is perhaps necessary to remind readers of the conditions under which this important work is carried out. The remuneration of a medical practitioner who serves the people in a prescribed area is £700 *per annum*, together with £50 house allowance. He also receives sixpence a mile travelling allowance. The practitioner is on duty during certain hours and is required to give medical attention to all the people in his area. If he is needed between his hours of duty he is allowed to make a charge of one guinea for each visit and of half a guinea for every attendance on a patient in his surgery. He is allowed to charge midwifery fees for patients who do not wish to go to the midwifery hospitals provided; he also has a month's holiday in each year, when his place is taken by another member of the Government service.

In our reference to Dr. Carruthers's report last year the suggestion was made that fuller information might be given, and in the report for the year ended December 31, 1939, recently received, this has been done. At the end of 1939 medical officers were engaged in the service of thirteen municipal districts. Most of the services had been in existence for the whole period of twelve months. In June, 1939, a medical officer was stationed in the Portland district, and in August, 1939, a second medical officer was stationed in the Scottsdale district, so that essential services might be provided at the North-Eastern Soldiers' Memorial Hospital, Scottsdale. The total population in the areas served by twelve medical officers is 23,106. The total number of attendances during the year, including those at the patients' residences, the surgery and hospital, workers' compensation cases and midwifery cases, was 23,090. Attendances at patients' residences numbered 9,226; attendances at the surgery numbered 12,741 and at hospital 739. The number of attendances in workers' compensation cases was 270 and in midwifery cases 114. The total mileage covered was 87,603. Last year we pointed out that the information then available did not enable us to estimate the average number of attendances per person *per annum*, and that there was no available information of the morbidity of people in Australia. From these figures it will be seen that each person in the districts concerned was seen on an average once during the year. This must not be taken as typical of all Australian communities, but as typical perhaps of sparsely populated country districts that are not industrial areas. In any attempt to make a true estimate of the number of medical attendances required by people living in country districts of this type some allowance would have to be made for those seriously ill persons who, once a diagnosis is made, are sent to larger centres where hospital accommodation is available for them. It will be seen that each medical officer made on an average 5·25 attendances a day, if Sundays and holidays are included. The mileage travelled varied considerably in different districts; in one area it was as high as 10,528, and in another as low as 2,697. On an average, however, each medical officer earned £183 during the year as an allowance towards the cost of mileage travelled. Altogether 9,226 visits were made to patients in their homes (hospital visits, 739, and attendances on midwifery cases, 114, are not included in this total) and the doctors travelled 87,603 miles to make them. This means that each attendance on a patient in his home meant a journey for the doctor of approximately 9·49 miles. These figures are of the greatest interest, and we are grateful to Dr. Carruthers for the trouble that he has taken to supply them. It is a

pity that no data are available for areas of other types, town or country. If Dr. Carruthers can devise some means of discovering the figures for medical attendances in some of the industrial centres of Tasmania, such as those on the west coast of the island, he will be doing a further service to the medical profession of Australia.

STUDIES ON PAIN.

PAIN plays so prominent a part in the life of man, and is so incompletely understood that any attempt to investigate its nature and control merits attention. An excellent device for producing pain the intensity of which can be accurately measured has been described by J. D. Hardy, H. G. Wolff and H. Goodell.[1] Using themselves as subjects, these workers have investigated certain aspects of pain production and control, submitting to the scrutiny of the laboratory the impressions fondly held by many clinicians with results that have a significance for both physiologist and therapeutist. The apparatus used consists of a 1,000-watt lamp, focused through a condensing lens and automatic shutter onto the forehead of the subject. The surface of the forehead is thoroughly blackened with Indian ink to ensure total absorption of the radiation and to prevent penetration of rays through the skin surface. The stimulus is thus made purely thermal, and its intensity regulated by a rheostat. The automatic shutter exposes the patient's forehead to the radiation for a test period of three seconds. The intensity of the radiation is gradually increased at intervals, and the level at which pain is first felt is noted.

All three subjects showed a remarkably constant "pain threshold" from day to day over a period of a year. Certain environmental factors were found to increase pain tolerance. Thus firm bandaging of the head raised the pain threshold by 4% to 6%; tight gripping of a bar by the hands caused a rise of 7% to 15%; a loud noise, intense enough to be "painful", close to the subject's ear caused a rise of 14% to 32%. Intense pain in the arm caused by occluding the blood flow with a sphygmomanometer cuff until at the end of an hour the pain was hardly bearable caused a gradual rise in the pain threshold as measured in the forehead to a maximum of 35%, corresponding in time to the maximum pain in the arm. We have all used counter-irritants at some time and know their value, and it is interesting to find this experimental corroboration of an impression that was undeniable. It is interesting, too, to find that exceedingly vigorous and painful counter-irritation increased the tolerance of pain by only 35%. Medicine in its crudest and most barbarous days could do as much as that.

Knowing, then, the limits of the witch-doctor, these workers proceeded to measure the relief afforded by the modern physician using various analgesic drugs. Morphine was the first drug investigated. Two of the subjects were given morphine in varying doses and one was given injections of sterile water. Each of the subjects was about the same weight, and all were healthy and free of pain at the time of the experiments. The control subject managed to raise his pain threshold by about 2% as a result of his injections of sterile water. The rise of threshold in the subjects given morphine was rapid and marked. The doses given varied from 0·5 milligramme (one one-hundred-and-twentieth of a grain) to 30 milligrammes (one-half of a grain), and the rise in pain threshold resulting ranged from 10% to 95%. The maximum analgesic effect increased in proportion to the dose of morphine from 0·5 milligramme to 15 milligrammes; but when a still larger dose was given the tolerance of pain showed little more significant increase. The duration of the analgesic effect also increased with increasing dosage, but the rate of increase with amount became progressively smaller, and increases of dosage above 15 milligrammes caused little significant rise in the duration of analgesia. The psychological effects of these doses of morphine make interesting reading. We must remember that all the subjects were keen persons with scientific analytical minds, and all were interested in the experiment being performed. They lacked perhaps the imagination of a De Quincey; they reacted in the way that we might expect of ourselves. Within three to four minutes of the injection the subjects became aware of feelings of muscle relaxation about the extremities, the neck and back. A few minutes later the mental attitude changed from one of alertness and concern about the experiment to one of contented apathy. Now there was no concern; all seemed to be going well. Intervals between readings, instead of being long and tiresome, were short and pleasant. This state of content lasted for two to three hours. After about half an hour loquacity developed, and mental concentration and logical thinking became increasingly difficult, a condition that lasted for about three hours. Vomiting was repeated and severe when the large dose of 30 milligrammes was given, but was associated with no more emotional reaction than would ordinarily accompany rinsing the mouth or swallowing. Lethargy persisted for as long as twenty-four hours. Towards the end of this period there developed impatience, annoyance and resentment, particularly if the subject was expected to engage in any deliberate mental activity. This was still difficult for him, but he was now conscious of his shortcomings and was sufficiently concerned to register annoyance.

The effect of morphine on prolonged pain was also investigated, and here the investigators came near to clinical problems. By means of screw clamps on muscles, constriction of the arm to impede circulation, immersion of a limb in ice-cold water, and distension of a balloon swallowed into the duodenum, severe and prolonged pain was produced. The striking fact revealed was that morphine was much more effective in controlling this pain if given some time (such as fifty minutes) before the onset of the pain. Unfortunately in practice the pain is usually present before the morphine is given; but it is plain that when morphine is given as premedication in preparation for a painful experience, adequate time should be given for the absorption of the drug before the onset of the pain. These experiments suggested that a little over one hour is a suitable time. By the end of that period the patient will be mentally apathetic and will have developed the maximum tolerance of pain from the dose of morphine given.

Two other observations of interest chiefly to physiologists were noted. The first was that perception of pain and heat are quite separate. The thresholds of heat and pain were measured by this apparatus. Then the subjects were each given 1·8 grammes (27 grains) of acetylsalicylic acid and the resulting changes in the threshold for heat and pain were followed over a period of four hours. The pain threshold was raised 35%, but the heat threshold was lowered 55%. In other words, these subjects became more tolerant of pain, but more acutely sensitive to heat. Moreover, when the circulation in the arm was occluded for a period of thirty-five minutes sensation of all types except pain was almost entirely absent in the hand. It seems plain, therefore, that heat and pain are subserved by quite distinct peripheral apparatus. The second observation was that when the area of stimulation was increased the pain threshold remained unchanged. Thus sensation of pain from a small area is just as acute as sensation of the same degree of pain from a large one. This does not mean, however, that the degree of discomfort and inconvenience to the subject is the same. Sunburn causes more discomfort if extensive than if localized to a small area.

This is interesting work of practical value. Many of the conclusions reached may already have existed as vague or as distinct impressions; but impressions are dangerous and unsatisfactory, and it is of the utmost importance to translate them to deliberately proven facts, as Hardy, Wolff and Goodell have done. This, however, is not the whole significance of their work. They have too developed a technique of pain mensuration which appears to be more accurate than any previously used, and have thus opened the way for more exact investigation of many aspects of this most important sensation.

[1] *The Journal of Clinical Investigation*, July, 1940.

Abstracts from Medical Literature.

SURGERY.

Latent Chorioncarcinoma.

A. F. BROWN, W. SNODGRASS AND O. B. PRATT (*The American Journal of Cancer*, April, 1940) report on the following case. A woman, thirty-two years old, entered hospital complaining of persistent cough with hæmoptysis of five months' duration. Examination suggested the presence of a tumour of the mid-section of the right lung. Surgical removal of this tumour was attempted, but was found to be impossible on account of the extension of the tumour into the mediastinum. The patient died soon afterwards, nine months after the onset of the symptoms. At autopsy a huge mass of red hæmorrhagic friable tumour tissue was found in the right lung. Numerous small tumours of the same kind were scattered through the rest of both lungs. There were metastases in the mediastinal lymph nodes and a large solitary tumour nodule in the upper part of the right kidney. Microscopic examination revealed a typical chorionepithelioma. The Friedman test carried out with extracts of the tumour tissue produced a positive reaction, although the tissue had already been fixed in formalin for twenty-four hours. A careful examination of the past history of the patient revealed the following data. The patient had had a normal pregnancy twelve years before death, and had had no abortions. Nine years before death her uterus was curetted for an apparent abortion of a two months' pregnancy. The material removed proved to be a hydatidiform mole. During the following time the patient had excessive vaginal bleeding and lower abdominal pain. Another curettage was done five months after the first. Two years after the removal of the hydatidiform mole, six years before the development of the chorionepithelioma, hysterectomy was performed. The case reported therefore presents the strange fact that the chorionepithelioma developed in the lung eight years after the hydatidiform mole and six years after the removal of the uterus. There arises the question whether benign chorionic tissue was embolized into the lung at the time of the hydatidiform mole, retained its vitality for eight years and then underwent malignant change. The other possibility is that a chorionepithelioma developed simultaneously with the mole and metastasized to the lung as a malignant tumour, then after removal of the primary growth from the uterus by curettage, the malignant deposit in the lung must have been held in an unusual kind of restraint during the long period of latency. The authors are not able to decide between these two alternatives. They stress the importance of their report as unusual proof of a genuine period of latency in this type of tumour.

Pain Following Cholecystectomy.

IT has been demonstrated that biliary pain can be produced by increased pressure within the bile ducts. J. M. McGowan and F. H. Henderson (*The New England Journal of Medicine*, June 6, 1940) have made use of T-tube drainage to decompress the ducts in the biliary tree and to study in detail the patency of the papilla of Vater by a series of pressure studies. If the usual indications are present, the common duct is opened during the operation, its patency is determined and an indwelling T-tube is inserted. Details are given of the post-operative care. After two weeks' time tests are carried out to determine the resting intrabiliary pressure and the perfusion pain level, and further radiological studies of the biliary tract are carried out. The normal resting fluid level is below 30 millimetres of water; readings of 30 to 100 millimetres of water suggest partial obstruction in the lower end of the common duct. This may be due to pressure from inflammation in the head of the pancreas, spasm of the musculature of the duodenal wall, a missed stone in the common duct, or œdema of the duodenal mucosa at the ampulla. Five deep inhalations of amyl nitrite or the sublingual administration of nitroglycerin, one or two tablets of $^1/_{200}$ grain, will aid in determining the nature of the obstruction, for a fall to normal pressure would indicate that spasm was the entire cause. It can be similarly shown that morphine increases spasm. Perfusion of the common duct is carried out in order to determine the amount of pressure that it can withstand without discomfort. Some patients experience pain when the biliary tree is exposed to relatively low pressures, at times not over 70 millimetres of water. This suggests that the bile ducts are dilated and high tension in the wall is produced by relatively low pressures, or the ducts are inflamed and unable to stand pressure. Normally the passages should be able to withstand a pressure equal to the maximum secretion pressure of the liver without the production of pain; this pressure is approximately that produced by a column of 300 millimetres of water. If pressures below this level produce discomfort, the patient is likely to suffer a recurrence of distress following removal of the T-tube. A series of radiological studies are also described following the slow injection into the T-tube of 10 cubic centimetres of "Diodrast". It is pointed out from these studies that a course of treatment with nitroglycerin will be of great value in biliary dyskinesia and also in some cases of cholecystitis. The use of morphine is limited to a diagnostic procedure, a typical attack of biliary dyskinesia being produced in a few minutes following the subcutaneous injection of one-sixth of a grain.

Dangers of the Use of Insulin and Glucose before Operation.

P. C. ESCHWEILER (*Surgery, Gynecology and Obstetrics*, August, 1940) deals with the dangers of the routine administration of insulin to cover intravenous infusion of solutions of glucose. The author quotes several cases among diabetics who required surgical or obstetric operations and who were given glucose by the intravenous and insulin by the subcutaneous route. The untoward results consisted of depression of the respiratory centre, as in post-operative collapse or collapse due to anæsthesia and in hypoglycæmic states evidenced by convulsions. From an analysis of the histories of a series of diabetics who were given glucose without any insulin prior to surgical intervention, the author deduces that following upon infusion of 500 cubic centimetres of 10% glucose solution even a patient with severe *diabetes mellitus* does not require the regular administration of insulin to bring the blood sugar values near to the fasting level. The author concludes that individual sensitivity to insulin varies very greatly and that insulin should never be given as a routine measure to "cover" glucose parenterally administered to patients with diabetes and that the blood sugar curve should always be estimated beforehand.

Gastroscopy.

H. J. MOERSCH AND W. WALTERS (*Surgery, Gynecology and Obstetrics*, August, 1940) present the results of their investigations of one hundred cases of gastric distress following upon operations on the stomach. The authors are satisfied that gastroscopy is very useful in the evaluation of symptoms which are frequently inconsistent with radiographic interpretations. The Wolf-Schindler flexible gastroscope makes it possible to obtain a highly satisfactory view of the interior of the stomach in most cases, especially if the details of the operation performed are available. In viewing the stoma more difficulty was encountered after gastro-enterostomy than partial gastrectomy. The findings in one hundred cases are tabulated according to the conditions found, under the following headings: "apparently normal mucosa", nineteen; "gastritis", twenty-eight; "erosive gastritis", six; "carcinoma", two; "benign tumour", one; "gastric ulcer", one; "gastro-duodenal ulcer", five.

Sulphanilamide in Renal Disease.

J. G. ALLEN (*The New England Journal of Medicine*, June 20, 1940) reports a case of wound infection after operation in a patient suffering from duodenal ulcer and advanced renal disease. The blood concentration of free and other forms of sulphanilamide rapidly became excessive, in spite of a dosage well within accepted limits. The excretion of sulphanilamide is similar to that of urea, and in this case the sulphanilamide was rapidly converted into the acetyl form. Detailed tables are given of the blood urea concentration, the level of chlorides in the blood, and the concentration of carbon dioxide in the blood during the progress of the illness. At autopsy a diagnosis of chronic ascending nephritis was made; little else worth mentioning was discovered.

The "Industrial Back".

R. T. JOHNSTONE (*The American Journal of Surgery*, June, 1940) protests against the loose use of the term "back sprain" in industrial medicine. A full investigation was made of over 3,000 such cases. In all the back pain had been considered to be due to the patient's work; but patients recently injured, with a definite history of severe falls, crushes and blows on the back, were excluded. Investigation included full clinical examination, the examination of X-ray films, examination of the urine and blood counts, and, when necessary, the performance of any other tests. In some 70% these investigations revealed some disease process which accounted for the pain, and in only 30% was it finally considered to be due to unusual strain or to strain in connexion with the patient's work. The author maintains that back pain is a problem for diagnosis and that disease and infection account for the majority

of the cases rather than unusual trauma. A statistical table is included, showing the enormous variety of conditions which were found in this long series of cases.

The Influence of Hot and Cold Applications upon Gastric and Intestinal Motor Activity.

J. D. ISGARD AND D. NYE (*Surgery, Gynecology and Obstetrics*, August, 1940) discuss the influence of hot and cold applications on gastric and intestinal motor activity. Normally the motor activity of the gastro-intestinal tract varies greatly from minute to minute and from hour to hour. The tract is acutely sensitive to widely varied influences, and the motor activity is stimulated or inhibited by such factors as the emotions, the reactions to the association with the senses of sight, taste and smell, the physical states of sleep and fatigue, and the direct factors of hunger and the ingestion of water and food. In the authors' experiments an inflated balloon was inserted into the ileum and connected to a manometer, and the changes were recorded. The tracings are reproduced in the article. Ice bags or hot-water bags were applied to the abdominal wall and to the thighs. Iced or hot water was given by mouth, and in addition gastric secretions were drawn off and analysed. The question of liberation of histamine in the circulation is discussed. The application of the findings to treatment in peptic ulcer, whether bleeding or not, and in inflammations of the alimentary tract is obvious. The authors find that gastro-intestinal motor activity is inhibited by the application of heat to the abdominal wall and by iced water taken by the mouth. It is stimulated by the application of ice to the abdominal wall and by the ingestion of not water by the mouth. External cold applications increase gastric acidity, increasing both free and total acids. By simply immersing the hands in iced water for a few minutes a response similar to that resulting from the subcutaneous injection of histamine can be obtained. The ingestion of iced water diminishes the secretion of acids by the stomach.

Gastric Tuberculosis.

RALPH C. SULLIVAN, NICHOLAS T. FRANCONA AND JACK D. KIRSHBAUM (*Annals of Surgery*, August, 1940) find that tuberculosis of the stomach is a very rare condition. This seems surprising in view of the high incidence of pulmonary tuberculosis, and especially as many of these sufferers have deficient gastric acidity. Tuberculosis of the stomach not associated with pulmonary tuberculosis has probably never been diagnosed prior to operation and is usually unsuspected even when the abdomen is open. Two pathological types of the disease occur—miliary lesions as part of a generalized infection, and ulcerative lesions in which are found numerous shallow irregular mucosal ulcers with overhanging edges and greyish-yellow bases, only rarely penetrating the muscle coat, but eventually in some cases causing scarring and shrinking of the organ and simulating syphilis or carcinoma. Eighty *per centum* of cases are of this ulcerative type. There may be no symptoms of the gastric disease. The authors report four cases, in only one of which were there any symptoms referable to the stomach. This patient complained that for six months he had experienced epigastric pain coming on during or immediately after taking food. It radiated to the back, lasted about one and a half hours, and was not relieved by the taking of food, alkalis, or by bowel actions. Starvation alone gave relief. There had been considerable loss of weight. Physical examination revealed a mass in the pyloric region, thought to be a carcinoma. X-ray examination supported this diagnosis. No free hydrochloric acid was found in a test meal. A partial gastrectomy was performed. Microscopic examination revealed the true nature of the process. Typical tubercles were found in the stomach wall and also in the regional lymph glands. The authors conclude from a review of the literature that tuberculosis of the stomach cannot be distinguished clinically from carcinoma or gastric ulcer, and may in many respects resemble gastric syphilis. As regards treatment, medical methods have proved of no avail. Resection whenever possible appears to be the treatment of choice.

Bilateral Intercostal Nerve Block for Upper Abdominal Surgery.

R. W. BARTLETT (*Surgery, Gynecology and Obstetrics*, August, 1940) describes a method of injecting the seventh to the eleventh intercostal nerves in the mid-axillary line, combined with light gas inhalation, for use on patients who are seriously ill and in conditions in which spinal and ether anæsthesia are contraindicated. Basal anæsthesia is used before the patient is moved to the theatre. A 2% solution of "Novocain" with three minims of adrenaline to the ounce is used, and forty cubic centimetres are injected in all; five injections of three to four cubic centimetres each are given on each side. The operations performed by the author were: exploration of the common bile duct, cholecystotomy, cholecystectomy, cholecysto-gastrostomy, biopsy of liver, aspiration of liver abscess, perforated typhoid ulcer, incisional hernia, umbilical hernia, transverse colostomy, secondary suture of the abdominal wall. No failures to obtain anæsthesia were experienced, and anæsthesia lasting for an hour and a half was obtained in one case.

Pheochromocytoma.

A SHORT discussion of the historical aspects and clinical features of pheochromocytomata is given by Alexander Brunschwig and Elanor Humphreys (*The Journal of the American Medical Association*, August 3, 1940). The clinical syndrome is apparently due to a sudden liberation into the circulation of an adrenaline-like substance secreted by the cells of this tumour, which occurs in or near the suprarenal gland. Sudden attacks of hypertension occur associated with headache, giddiness, weakness, tachycardia, precordial pain and maybe syncope. The attacks may last several moments to several hours. They usually increase in severity and may be fatal. Between the attacks the blood pressure may be normal. A case is reported. The patient had attacks of such severity that in one life had been pronounced extinct, but she later revived. In a subsequent attack the systolic blood pressure rose to 295 and the diastolic to 155 millimetres of mercury. Pheochromocytoma was diagnosed and such a tumour was demonstrated at operation in the right suprarenal area and was excised. Over twelve months later the patient remained well. The systolic blood pressure was 100 and the diastolic pressure 70 millimetres of mercury. For operation the authors use the abdominal route. It is pointed out that the tumours may be bilateral and may occur entirely outside the adrenal gland. These points and the difficulty of localizing the tumour prior to operation make the abdominal route preferable to the lumbar.

Fæcal Fistula.

ARTHUR N. COLLINS (*The Western Journal of Surgery, Obstetrics and Gynecology*, August, 1940) discusses fæcal fistula after appendicectomy, a distressing, disabling and even fatal complication. He reports a series of eighteen cases following appendicectomy, and gives details of the conditions found at the primary operation and the procedure carried out at that time. All were cases of a serious nature, in which gangrene of the appendix or abscess formation had occurred. Fistulæ appeared from the third to the twenty-fifth day and lasted for from two weeks to two and a half years. After examining the records the author goes on to deprecate the practice of simply tying off the stump and leaving it free and not inverted. He points out that there is nothing to resist gas pressure in the caecum except a frail ligature which easily softens and slips off, especially in the presence of infection, and thus leaves an open hole in the caecum. He feels that although the difficulties may be great, the gut must be closed and serosa approximated to serosa. In addition, tension in the gut must be relieved.

The Intraperitoneal Use of Hypertonic Glucose Solution in the Prevention of Adhesions.

H. P. TOTTEN (*Surgery*, September, 1940) presents the results of an experimental study undertaken to determine the value of the intraperitoneal introduction of hypertonic glucose solution in preventing the formation and reformation of experimentally produced adhesions. Rabbits were used for the experiments. It was found that 20% glucose in normal saline solution is well tolerated in the peritoneal cavity. An equivalent amount of normal saline solution was given subcutaneously to obviate dehydration. Adhesions were produced by combined mechanical and chemical trauma (scraping with a knife blade and painting with tincture of iodine). In two parallel series of sixteen animals each adhesions were produced in all the control animals, while 14 of the animals which received the hypertonic solution formed no adhesions. Of the two rabbits in which adhesions formed, the condition in one was complicated by wound infection, and in the other the adhesions were slight. In 12 animals of the second group further trauma unaccompanied by glucose solution led to adhesion formation in all cases. Finally, at reoperation, in 11 of the control animals adhesions were separated and glucose solution was introduced, with the result that in seven adhesions did not reform. The author concluded that preliminary glucose injections induced a degree of non-specific immunity in the peritoneum, but that in the presence of gross peritoneal contamination hypertonic solution by prevention of adhesions hastens the spread of infection.

Special Articles on Psychiatry in General Practice.

(Contributed by request.)

XXIV.

COMMON PITFALLS IN TREATMENT.

THE old proverb runs that "to err is human". It is countered by another that the wise man profits by his mistakes. Some are obvious, others are obscure, but all are important.

The commonest mistakes are those caused by forgetting the power of suggestion. The individual's capacity to absorb information is rightly considered a fundamental instinct. We are all highly suggestible. We absorb ideas as readily as ink flows to a blotting paper, or as germs invade an open wound. The technique of suggestion in psycho-therapeutics should be as meticulous as that of asepsis in surgery. This will be obvious if the following examples are carefully studied. Remember that the mental invalid is peculiarly suggestible.

Never Discuss a Patient in the Hearing of Another Patient.

Whilst this may be a counsel of perfection, it should be followed whenever possible, if only to prevent such a catastrophe as follows:

Mrs. X, in the wards of a general hospital, was making a satisfactory recovery. A physician demonstrated a case of cancer in the adjacent bed. He outlined the course of the disease, and inadvertently one of the students let his glance fall on Mrs. X. It was sufficient to demonstrate to her that the talk was about her condition. She had cancer. The long arm of coincidence gave her an obstinate pruritus. It was "obviously cancerous". In spite of all reassurance she left hospital worse than she entered—a recruit to the army of cancerphobiacs.

Never Forget that a Seemingly Inaccessible Patient May have Insight.

Often patients give an appearance of such complete apathy that they may be considered incapable of understanding. They are traps for the unwary.

Mrs. A used to sit in the corner of a ward. Skirt over her head, head and shoulders bowed, she seemed a classic picture of disinterest. Actually she remembered everything within earshot and on her recovery was with difficulty prevented from exposing the hospital gossip to a journal famed for sensationalism.

Remember that Many Mental Invalids have Hyperacusis.

Probably the commonest mistake of all arises from not appreciating the extraordinary hearing capacity of some neurotics. They have what amounts to a sixth sense. *Sotto voce* remarks that certification or a mental hospital may be necessary, or even the mention of such words as insanity, delusion, hallucinations, gives the patient a belief in hopelessness. It is literally true to say that many grave psychoses have arisen from this cause.

Give Your Grave Prognoses only to Tactful Relatives.

The keystone of successful psychotherapy is optimism. A hint of pessimism may do harm that cannot be undone by an hour of optimism. The physician, in order to be strictly truthful and save his prestige, often feels compelled to stress the dangers. Remembering that the welfare of the patient is the sole objective and that friends tattle, he must remember to coat the pill of truth with the covering of optimism. His prestige is a secondary matter.

The Infection of Doubt.

There is excuse for doubt, since seldom are patients cast-iron therapeutic certainties. Doubt may be indirectly expressed through an ill-planned clinical examination. The physician, for example, does a gastric analysis, later takes an X-ray photograph and then follows it up with a blood analysis. The patient scents indecision and diagnoses doubt. Another method of instilling doubt is by the use of such words as "I think", "I hope", "I trust" *et cetera*. The physician should shun such expressions.

Transference is a Double-Edged Weapon.

Whilst it is essential that the patient should have faith in his doctor, there are pitfalls in this regard. Transference is often on the social level. The patient regards his medical adviser as a friend. This is particularly the case of the general practitioner. When symptoms such as fear of suicide, insanity or homicidal impulses occur, the patient refuses to discuss them because he fears the loss of prestige. The eradication of this tendency is difficult. The practitioner must sense the existence of an inhibition and insist that the patient have a consultation with a psychiatrist. Often one interview will be sufficient to show that the symptom has no significance and the road to good health is easy.

The Family History Pitfall.

Text-books of psychiatry pay great attention to the family history. They show that much mental invalidism is inherited. There is every excuse for a discussion with the patient on this aspect of his disorder. A little reflection will, however, show that even if a morbid family trait is discovered, it will not help in treatment. One's ancestors are truly beyond recall or alteration. The discovery will give to the patient a sense of hopelessness. He must realize that the dice are heavily loaded against recovery. In order to obviate this it is wisdom in a therapeutic sense to ask no direct questions from the patient as to the family tree. A few discreet investigations from the family and friends will provide the necessary data to aid in the prognosis.

The Bugbear of Suicide.

That all depressed patients are potentially suicidal produces great difficulties in treatment. Whilst every precaution must be taken, common mistakes occur from the following causes: (*a*) Failure to realize that the most dangerous period is when the patient has almost recovered. (*b*) Over-suggestion of means to prevent suicide, thereby suggesting to the patient that he is likely to end his life. (*c*) Sending depressed patients on sea voyages, thereby placing in his grasp an easy means to *felo de se*. A leap overboard is a time-honoured escape from reality.

The Misuse of Hobbies.

Whilst occupational therapy is a most valuable aid to recovery, it has many pitfalls. Its aim is to promote mental concentration and produce a one-track mind. The best means to this end are by creative effort in something difficult to accomplish, and there must be an element of interest. Common mistakes result from not realizing that hobbies such as reading and music actually produce a many-track mind. They are effortless and non-creative. To prescribe sport is useful; but it must be remembered that sport can be enjoyed only in daylight. It is essential to find occupation in the evenings and the daylight hours, when sport is out of the question. To prevent errors the only efficient plan is to regard occupation as a prescription. Every detail must be thoughtfully considered with the goal of occupation for each hour of the day. Short cuts and generalities are as useless as in any other method of treatment.

The Magic Holiday.

Undoubtedly mental invalidism is often a result of environment. If we change the locale we break the vicious circle and promote recovery. Too often the holiday prescription is issued far too early. The patient has to return to his environment. He recovers for a while and then repeats the error of his ways. There is a relapse. The only method of preventing this is to discuss fully with the patient all the details of his environment. Show him how his mode of living can be altered. Then send him away to meditate on his good resolutions and when he returns see that he fulfils them. The real therapeutic effort must precede the holiday, and the holiday must be postponed until the effort is accomplished.

Euphoria is but an Expression of a Balanced Life.

It cannot be too frequently stressed that human life is a complicated balance between many forces. There is the physical body. There is the mind and the environment, which constantly impinges upon both body and mind. Euphoria can exist only when all are in harmony. Unfortunately we physicians are not immune from bias. We become psychologically minded and see life in the mirror of mind. Or we may worship at the shrine of the test-tube. On occasion we may see life from the angle of the social crusader and magnify the factor of environment. Our viewpoint may vary from day to day at the suggestion of the Press, our colleagues or even the traveller who introduces us to new drugs. We are indeed devotees at the altar of fashion. It is this very changeability which produces many

of our most obvious mistakes. We see the physical derangement of our patients and gloss over the equally important psychological factor or *vice versa*. Perhaps we ignore altogether the environmental factor which is undermining our attempts at cure. To avoid such fundamental errors there must be strict observance of a simple rule. The physical, the psychological, the environmental must be assessed in their true proportions and must be corrected.

Time is our Greatest Ally.

There is a proverb that the wise man profits by studying his enemies. In this connexion the quack provides a useful lesson. He flourishes not by reason of his skill, but by his belief in the efficacy of time. Admittedly it is difficult to listen to the timeless vapourings of mental invalids. They sap the energy of the listener and create an atmosphere of irritability. But if one's aim is to prevent mistakes and promote cure, they must be endured. It is probable that more patients have been lost by impatience than by any other single means.

Conclusion.

This homily on mistakes in therapeutics commenced with the aphorism that to err is human. Our goal must be the minimum of errors. We shall achieve this only by attention to detail. Every word, every test, every prescription must be considered as part of a general plan. One weak link will ruin the integrity of the chain. The physician has to be on ceaseless guard, realizing that he is dealing with conflicting intangible yet important realities. The key words are patience and foresight. With them he will make some mistakes. Without them he will reap a veritable harvest of errors.

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British Medical Association News.

SCIENTIFIC.

A MEETING of the South Australian Branch of the British Medical Association was held on September 26, 1940, at Adelaide.

The Control of Tuberculosis.

DR. D. R. W. COWAN read a paper entitled "Control of Tuberculosis" (see page 627).

DR. H. W. WUNDERLY first of all stressed the importance of the investigation work done at the chest clinic. He said that the Government, recognizing its value, had made it possible for any case to be thoroughly investigated without cost to the patient. Thus the "suspect" and the medical attendant had the benefit of the results of the skin test and examination of the sputum, and of a report on the X-ray findings in the lungs. At the present time the service was limited more or less to the examination of contacts and of patients of private practitioners who were referred for investigation. The work was so important that Dr. Wunderly suggested that it be extended to two further groups of people—all the unemployed who were receiving rations and all women attending antenatal clinics.

There were many children in the community who would benefit by a few months in a preventorium. Why was it not possible to make those in authority realize that prevention was better than cure? Preventoria had been tried in the United States of America, and the present tendency was to remove the infecting parent from the family rather than the children away from the home. It was universally recognized that most children recovered from the primary infection if the chain of infection was broken.

Dr. Wunderly was in complete agreement with Dr. Cowan that it was not a good plan to return a man, when his disease had been arrested, to the sphere of life and work in which he originally broke down. An industrial colony was expensive, and it needed much careful planning and capable administration; but it was worth all the effort. The war against tuberculosis would never be won if the plan of campaign was to be directed by the mentally lazy.

It was generally agreed that prognosis was related to the stage at which the disease was detected. In the fifteenth annual report of the Ministry of Health it was pointed out that there had been no increase in the proportion of new cases of pulmonary tuberculosis reported to the number of deaths from the disease; this suggested that there had been no increase in the proportion of cases reported in the early stages. It was humiliating to be told by D'Arcy Hart in his Milroy Lecture that, except in the small group of patients who could be treated by collapse therapy, prognosis had not been materially affected by advances in treatment in the past thirty years. Hart insisted that what was required was a procedure whereby progressive lesions might be discovered in the asymptomatic or preclinical stage and as early as possible in their development. Surveys conducted in other parts of the world, more particularly in the United States of America, had been the means of detecting many lesions in the preclinical stage, and as a result the prognosis for the individual patient had been materially improved. In addition, as had been shown in the Framingham demonstration, to which Dr. Wunderly would refer later, the tuberculosis death rate was quickly reduced. Further, patients suffering from the disease were detected before they were infectious, and so they were prevented from spreading infection. Three large surveys in the United States of America and the survey conducted with the aid of a grant from the National Health and Medical Research Council, showed that approximately 2% of the working population had active pulmonary tuberculosis. When the figures for the various fighting forces were collected it would be found that, in spite of two or more medical examinations, something over 1% of the recruits were found to have radiological evidence of definite pulmonary tuberculosis. Framingham had a population of 17,000, and the demonstration was made possible by a grant from the Metropolitan Life Assurance Company. The percentage of cases discoverable in the early stages increased from 55 to 88, while that of cases reported before the patient's death increased from 60 to 93. At the time the demonstration began (in 1917) the tuberculosis death rate was 97·5 per 100,000; in 1938 the death rate was 17 per 100,000. In 1937 the death rate for South Australia was 38·19 per 100,000, the highest but one in the Commonwealth. The Framingham demonstration also showed that there were nine persons suffering from active pulmonary tuberculosis for each annual death, and that in addition there were at least an equal number of persons with latent or quiescent lesions. Probably under present conditions it was possible to discover seven persons with active lesions for each annual death, provided that all possible case-finding facilities were utilized.

Dr. Wunderly then discussed case-finding facilities, and stressed the importance of cultivation of the tubercle bacillus from sputum and the product of gastric lavage. He quoted directly from a letter that he had received a few days previously from Dr. Reginald Webster, as follows:

> During recent months I have detected tubercle bacilli in 53 subjects of P.T., the majority being young adults.
>
> The 53 positive results are made up as follows:
>
> 20 by direct smear examination of sputum.
>
> 17 by culture from sputa in which the smear examination failed to detect Tb.
>
> 16 by culture from mucus aspirated from the fasting stomachs of patients who could not produce any sputum.
>
> Thus by the adoption of cultural methods and the utilization of gastric contents the number of positive results is more than 2½ times the number that would have been obtained had the examinations been restricted to Ziehl-Neelsen smears.
>
> I have worked out two groups as follows, with bacteriology complete and correlated with the X-ray findings:
>
> *A.* Classed as radiologically active: 54, of which 33 positive—61%.
>
> *B.* Classed as radiologically inactive, as old and healed: 40, of which 5 were positive—12½%.
>
> Combining the two—38 positive results in 94—40·4%.

Dr. Wunderly went on to say that Dr. Webster's findings had an important bearing on what he (Dr. Wunderly) wished to say with regard to Dr. Cowan's comment on the radiological examination of recruits for the Australian Imperial Force. The interpretation of the shadows was not quite so odd as Dr. Cowan had suggested. Up to the present, in their particular military district, the standard had been to accept those recruits who presented radiological evidence of a healed primary complex—a calcified area in the periphery of the lung field, with calcification in the corresponding hilar gland. Dr. Wunderly knew perfectly well that some of these areas of calcification were harbouring living tubercle bacilli, and that some of these lesions, under active service conditions, would become active; thus some of these recruits might develop an endogenous reinfection.

On the other hand, these recruits, who had successfully dealt with their primary infection, had an increased immunity and so an increased resistance to an exogenous infection. All recruits with radiological evidence of secondary reinfection or adult type of pulmonary tuberculosis were rejected. Those with "pleural caps" or very fine spots of calcification at the extreme top of the pleural shadow were not necessarily classed as tuberculous, and these recruits were accepted. Dr. Cowan appeared to think that the swimmer and Rugby footballer to whom he had referred in his paper and who had small lesions above both clavicles, had been harshly treated and should not have been rejected; but Dr. Cowan's own series of cases showed that this was a dangerous area and that people with very small apical lesions could produce sputum containing tubercle bacilli; this fact was supported by the results of Dr. Webster's research.

Thus in addition to the methods of tuberculosis control suggested by Dr. Cowan (the provision of more bed space, and the establishment of an industrial colony and of a preventorium), Dr. Wunderly advocated a more thorough search for *Mycobacterium tuberculosis* and the carrying out of mass surveys to detect the disease in its preclinical stage. The radiological examination of the members of the Australian Imperial Force, of the Royal Australian Air Force and of the Royal Australian Navy was such a survey, and a wonderful opportunity would be missed if such an examination was not made of the members of the militia. The Repatriation Department admitted that the average cost of each case of pulmonary tuberculosis since the war of 1914-1918 was £3,000. Quite apart from the question of finance, the reduction in the number of infectious cases amongst the fighting forces would have a very beneficial effect on the health of the men. Dr. Wunderly said that if they were going to do nothing but wring their hands helplessly and say that the problem of tuberculosis was a terrible thing, but they were going to do nothing further about it, then they might as well abandon the ideal of national fitness. That campaign would achieve very little if they were not going to deal with such an active "fifth columnist" as the tubercle bacillus. In order to start a discussion, Dr. Wunderly said that it was better to detect persons suffering from tuberculosis and to have no beds in which to put them than to leave them undiscovered amongst the community; the "undiagnosed case" was the most dangerous.

Dr. Cowan, in reply, thanked the members for their kind reception of his paper. He realized that some "contacts" would refuse examination from fear either that something would be found wrong with them or that their jobs might be jeopardized; but he considered that voluntary methods were preferable at present. Dr. Wunderly had suggested a survey of the whole community, even though facilities for the care of patients found might not be available. Dr. Cowan opposed this view and considered it necessary that provision for the care, both medical and economic, of patients discovered, should go hand in hand with case-finding efforts. Mass surveys must be either on a compulsory or on a voluntary basis. If the surveys were compulsory it would be unfair merely to inform sufferers of their trouble and to seek their help in preventive measures without providing them with the means for proper care and treatment. If the survey was on a voluntary basis, people would soon cease to volunteer unless facilities for proper care were provided, and the whole scheme would quickly fall into disrepute.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held at the Children's Hospital, Carlton, Melbourne, on July 10, 1940, DR. H. DOUGLAS STEPHENS, the President, in the chair.

"Clicking Knee Joint."

DR. RUSSELL HOWARD showed a boy, aged twelve years, apparently in robust health, who had a clicking knee joint of considerable interest from the point of view of diagnosis and treatment. In November, 1939, it was ascertained that the boy fell out of bed, landing comparatively gently on his left knee without any twisting movement. The knee was sore for a few days, but it was not considered necessary to consult a doctor. One month later a clicking was noticed, which had persisted; it was unaccompanied by pain, swelling or disability. The child was brought to the out-patient department merely because of the clicking, which could be elicited at any time without difficulty. On questioning him closely Dr. Howard ascertained that occasionally he had some difficulty in carrying out complete extension at the knee joint; but the difficulty was overcome easily and he had no pain or swelling afterwards. Dr. Howard said that the trouble appeared to be placed medially and anteriorly in the joint; it was intraarticular and occurred just short of full extension. He added that it could be obliterated by lateral pressure from the medial aspect when traction was applied to the leg. An important additional discovery was that at each of two levels measured by Dr. Howard wasting was present in the quadriceps muscle of the thigh, represented by a circumferential difference of one and a quarter inches; the wasting could not be due to disuse atrophy, because the boy had enjoyed a full range of movement at the knee joint and had been normally active.

Dr. Howard expressed the opinion that there was something wrong with the lateral meniscus; he thought he could feel some abnormality between the lateral condyles of the tibia and the femur. The clicking could always be related to the same site. There was not a classical cyst of the lateral meniscus; but a congenitally hypermobile lateral meniscus might have become somewhat displaced. With reference to treatment, Dr. Howard was of the opinion that something should be done, as he regarded the wasting of the quadriceps as a distinct indication. He proposed to open the knee joint rather widely by splitting the patella lengthwise, and he said that he would show the patient again at a later meeting.

Intrathoracic Tumour.

DR. J. W. GRIEVE showed a girl, aged nine years, who had a tumour, abscess or cyst in the right side of the chest, which for twelve months had led to recurrent attacks of pain and limitation of movement of the right side of the chest. In December, 1939, she had been acutely ill, with a high temperature and some rigidity of the neck; meningitis was suspected, but the illness was of brief duration. At the time there was some coughing, but no sputum or blood was produced. In April, 1940, she was admitted to the Geelong Hospital suffering from pain in the chest, shortness of breath and delirium. It was thought that she had pneumonia at the right apex. One week later, as dulness to percussion had continued after the acute signs subsided, an exploratory needle was introduced into the dull area over the right side of the chest. Fifteen cubic centimetres of straw-coloured fluid were obtained, which contained a few leucocytes, and which failed to yield any organisms when culture was attempted. From that time the signs had not subsided and the temperature was usually somewhat elevated. Towards the end of May, as the chest was becoming increasingly bulged and the position of the apex beat had reached one inch outside the nipple line, the presence of a hydatid cyst was suspected, though there was no evidence that the child had been at any time exposed to infestation from dogs. The Casoni intradermal test was performed and a positive reaction was obtained. Dr. Grieve said that at that stage the child was admitted to the Children's Hospital. The temperature was not raised and she looked well; but examination of the chest revealed local bulging on the right side and very poor mobility. With the exception of a small area at the right apex and a slightly larger area at the base, the right side of the chest was very dull to percussion. The breath sounds were not audible over the dull area, but were tubular at the apex and high-pitched at the base; no adventitious sounds were heard. Grocco's triangle was mapped out over the base of the left lung. The position of the apex beat of the heart was in the sixth left intercostal space, one and a quarter inches outside the nipple line. The heart sounds were normal. The edge of the liver was palpable two centimetres below the right costal margin.

In his description of the investigations which had been carried out, Dr. Grieve said that the child's blood serum had failed to react to the hydatid complement fixation test; on two occasions the Casoni intradermal test had produced no reaction, and only 2% of the leucocyte cells were eosinophilic. In the skiagrams the appearances were suggestive of the presence of a hydatid cyst; a large homogeneous mass could be visualized bulging postero-anteriorly and laterally, and pushing the trachea over to the left side; but the diaphragm was seen to be free of the mass. From a bronchograph it was estimated that the large rounded mass occupied four-fifths of the right side of the chest and prevented filling of the right bronchi except towards the base, where the lung was compressed. The mass extended beyond the mid-line, compressing the trachea and to a certain extent the left lung, and displacing the mediastinal contents to the left. The third rib on the right side was eroded, but it was difficult to form an opinion as to

whether the tumour arose from it; the erosion might be due to pressure.

Dr. Grieve went on to say that there was no clinical evidence of pressure on the mediastinum, though the patient was slightly dsypnœic and the superficial veins of the chest were perhaps unduly prominent. The problem was to decide the nature of the swelling. He was anxious to obtain opinions as to whether it arose from the lung or from the mediastinal contents, and asked for suggestions as to further investigation and suitable treatment. The patient had been afebrile while in hospital, and as the presence of the tumour had been evident for a year he did not think that it was malignant.

DR. J. G. WHITAKER drew attention to the indications in the skiagrams that the tumour was placed anteriorly and that the rib erosion was posterior. He said that he did not think they could discard the diagnosis of chronic empyema. At all events, a very large exposure would have to be made if surgical intervention was undertaken. He was also of the opinion that the thyreoid gland was enlarged.

DR. KEITH HALLAM said that the case presented a difficult radiological problem. The radiolucent area at the base of the right lung might be either intrathoracic or intra-abdominal (pneumoperitoneum). The right half of the diaphragm was well above the level of the left, and the radiolucent area was below it. It was quite possible that multiple hydatid cysts were present and that one or more of them might have been ruptured; the child had had recurrent pain and dyspnœa and there had been febrile episodes. Dr. Hallam thought it worth while to mention pneumoperitoneum because he had known it to simulate spontaneous pneumothorax so closely as to delude even the cleverest of physicians. He suggested that further plain postero-anterior and antero-posterior skiagrams should be taken of the abdomen to make sure of the position of the lower level of the liver, in order to do all that was possible to exclude the presence of an intraabdominal cyst.

DR. W. MCL. SMITHERS said that as the child was neither toxæmic nor cachectic the performance of artificial pneumothorax for diagnostic purposes was feasible. The lung would be compressed and its shadow thrown into relief; and if the pneumothorax was continued to the time of operation the surgeon might have better access to the site.

DR. H. BOYD GRAHAM said that as exploration had been carried out on a previous occasion without misadventure, and as there was still a strong suspicion of the presence of at least one hydatid cyst, he thought that, with adequate safeguards, aspiration of a little more fluid should be made for diagnostic purposes. The fluid so obtained should be tested for hydatid antigenic qualities and also for the other characteristics of hydatid fluid. When multiple cysts were present which had leaked or had had other accidents, it was usual to obtain fixation of many units of complement for a long period of time, running into years.

DR. GUY SPRINGTHORPE suggested that possibly a previously existing empyema cavity had been sterilized by means of sulphapyridine therapy, and that the result was an encysted lesion containing sterile pleural fluid.

PROFESSOR R. D. WRIGHT expressed the view that the performance of a quantitative Aschheim-Zondek test might throw light on the likelihood of the presence of a dermoid cyst.

DR. H. DOUGLAS STEPHENS said that if the tests were adequate the presence of hydatid cysts could be excluded. In that event he considered that the possibilities were congenital cysts of the lung, chronic cysts or empyema.

Magnesium Metabolism.

PROFESSOR R. D. WRIGHT gave a short address on the subject of magnesium metabolism. With reference to magnesium deficiency in laboratory animals he mentioned vasodilatation in the skin and hyperexcitability, and said that if the deficiency was extreme a form of tetany developed from which recovery was unusual. He stated that in less acute deficiency the symptoms were hyperæmia of the skin, and especially of the mouth, leading to sponginess of the gums and affecting the extremities; the occurrence of exudative dermatitis had been noted; and the animals frequently had hyperreactivity to stimuli. He added that following the onset of those symptoms, poor appetite, loss of weight and malnutrition might occur.

Professor Wright briefly discussed the circumstances in which magnesium deficiency had been recorded as occurring more or less naturally. In the condition called "grass staggers" in cows, the cow developed tetany when placed on grass feed after being stable-fed for the winter and giving birth to a calf. Calves might develop magnesium tetany when reared on whole-milk feeding without forage. There were records of magnesium deficiency in human subjects who were fed on a hospital "soft diet" for long periods. Young rats had been known to develop symptoms of magnesium deficiency when suckled by mother rats receiving a dietary containing a basal amount of magnesium.

DR. H. BOYD GRAHAM, at the request of the Chairman, thanked Professor Wright for the information he had given on a novel ubject of discussion at the meetings of the society. It was apparent that the clinical application of the information would require a considerable amount of study; but Dr. Graham was glad to know that Professor Wright had consented to be associated with the clinicians in that work.

Hypertrophic Pyloric Stenosis.

DR. HOWARD WILLIAMS presented a review of the research work he was conducting on the problem of congenital hypertrophic stenosis of the pylorus. He referred to work published in THE MEDICAL JOURNAL OF AUSTRALIA on March 18, 1939, at page 414. He reported that the investigation had been continued and was still in progress. Dr. Williams had satisfied himself that a sufficiently large number of infants had been treated in the way he had outlined in his previous communication to justify his assertion that the adoption of the method reduced the mortality rate. Before subjecting the infant to any surgical measures he corrected the disordered biochemical processes, and the nutrition of the infant was the subject of special care after the operative procedure.

Dr. Williams announced that 60 infants had been treated and the mortality rate of the group was only 5%; that figure was considerably lower than the 16·8% mortality rate for 250 infants treated in the ten-year period from 1927 to 1937, immediately preceding the commencement of the research work. He added that the risks of operative measures had been minimized and the post-operative courses were characteristically free from sudden collapse and the other alarming manifestations which had occurred not infrequently in the period prior to the introduction of the new method of treatment. He expressed the hope that it would be possible to continue the work; he was anxious to attempt to define with exactitude the role of antispasmodic drugs in treatment. In conclusion, Dr. Williams acknowledged his indebtedness to Miss Green, biochemist to the pathological department at the hospital, who had carried out the biochemical analyses.

DR. J. G. WHITAKER thanked Dr. Williams for his report on what Dr. Whitaker believed to have been the best original work done at the hospital for many years. The figures that Dr. Williams had given concerning the reduction of the mortality rate spoke for themselves. Dr. Whitaker could testify that it was a decided advantage to operate on normal children instead of on children who were dangerously ill. He remarked that the technique of treatment by the intravenous administration of saline solution at the Children's Hospital was far ahead of that of other hospitals in Australia; that was due to the enterprise and initiative of Dr. Howard Williams and of the preceding medical superintendent, Dr. Vernon Collins. Their energy had been most inspiring. The well-directed effort Dr. Williams had put into the research work was most praiseworthy.

DR. J. W. GRIEVE added his congratulations to those of Dr. Whitaker. Dr. Grieve had felt that radiographic investigation was not of very much assistance to him in the diagnosis of hypertrophic pyloric stenosis. He was also interested in the more accurate definition of the group of patients suitable for treatment by antispasmodic drugs; that accuracy might emerge if Dr. Howard Williams proceeded with the research.

DR. GUY SPRINGTHORPE also congratulated Dr. Williams on the work he had done. He wondered whether Dr. Williams would advise the estimation of the urinary chloride content as a single test as to whether a child's biochemical state was suitable for successful operative interference. He remarked that Dr. Leonard Findlay had reported findings in the radiological investigation of pyloric stenosis so variable that they depreciated the diagnostic value considerably.

DR. H. DOUGLAS STEPHENS expressed surprise that at the hospital they were able to keep the barium in the babies' stomachs sufficiently long to obtain a record of the residuum after four hours; in work amongst private patients the risk of early vomiting of the barium vitiated the value of the test as an aid in the differential diagnosis.

DR. J. H. COLEBATCH also congratulated Dr. Williams on the thoroughness and usefulness of the work. The intravenous administration of saline solution had been used at Great Ormond Street Hospital, London, for a number of years in treatment and in preparation for operation for pyloric stenosis. Dr. Colebatch believed that Dr. Bateman

had used it in routine fashion there since Dr. Ian Wood had introduced it when he visited London in 1932. When Dr. Colebatch was in London in 1937 saline therapy was in general use. In the clinic of Dr. Donald Paterson they had had approximately 50 cases within six months, and the only death that had occurred was one some two months after operation. It was not to be forgotten, however, that in London they had a newly built hospital with excellent facilities for nursing the patients to reduce the mortality rate from infection. Dr. Paterson insisted that no more than three nurses should handle any one baby. He believed that at the Vincent Square Hospital for Infants the mortality rate was between 1% and 2%, though the rate was slightly higher at Great Ormond Street. It was usual to send the patients home on the fifth day after operation, with instructions to the parents to return on the eighth day for removal of stitches and to attend regularly for supervision of the diet. The barium meal investigation was used as a diagnostic aid in London; but Dr. Colebatch did not think that it was of much assistance in doubtful cases. The intervention of spasm could account for delay in the emptying of the stomach. At the time he was in London he had not known about Dr. Williams's method of controlling the intravenous therapy by biochemical estimation. "Eumydrine" had been used successfully in only two out of the 50 cases of pyloric stenosis; Dr. Colebatch had heard that the initial enthusiasm for "Eumydrine" was diminishing considerably.

Professor Wright also congratulated Dr. Williams, who was carrying out a life-saving piece of work on applied physiology. Professor Wright pointed out that there was more in the problem than the simple loss of chlorides; the child was secreting a fluid with a high content of hydrochloric acid, and through vomiting was losing the hydrogen ions as well as the chloride ions that ordinarily bound the base. He did not know how the body could be made to replace the hydrogen ions readily. It was known that the stomach would go on stealing chloride until the body supply was nearly depleted; the kidneys knew when to stop, but the stomach did not. At the University of Melbourne there was a perfect specimen of pyloric stenosis obtained at the abattoirs from a pig which had been fattened as a porker; the capacity of the undistended stomach was approximately one gallon. If a stomach passed the phase of acute blockage of the pylorus, it might come to the stage of organic obstruction and extreme hypertrophy.

Dr. Williams, in reply, expressed his appreciation of the interest in and approval of his work. He thought that the presence of much chloride in the urine could be regarded as a justification for deferring operation. He doubted whether radiographic evidence of gastric retention should be regarded as an indication for operation, but he believed that if the stomach emptied through the pylorus within four hours, the patient would not benefit from operative interference.

The Royal Australasian College of Physicians.

EXAMINATION FOR MEMBERSHIP.

THE next examination for Membership of the Royal Australasian College of Physicians will be held in March and April, 1941. On this occasion, for the convenience of candidates, the clinical portion of the examination will be conducted in both Sydney and Melbourne.

By-law 10, Section II, states:

> The Examination for Membership of the College shall consist of:
>
> (1) A paper on the Principles and Practice of Medicine including Pathology, Therapeutics and the History of Medicine.
>
> (2) An oral examination, which may include the clinical examination of patients, together with the identification of naked-eye and microscopic specimens.

The dates upon which the examination will be held are as follows:

> Written examination (capital cities): Saturday, March 8, 1941.
>
> Clinical examination (Melbourne): Saturday, March 29, 1941.
>
> Clinical examination (Sydney): Wednesday and Thursday, April 2 and 3, 1941.

Application forms may be obtained from the office of the College, 145, Macquarie Street, Sydney, and should be in the hands of the Acting Honorary Secretary at this address not later than February 8, 1941.

A further examination will be held in August and September, 1941, full details of which will be announced at a later date.

Obituary.

HOWARD WOODRUFF LORDING.

WE regret to announce the death of Dr. Howard Woodruff Lording, which occurred on November 3, 1940, at Hawthorn, Victoria.

Diary for the Month.

DEC. 17.—New South Wales Branch, B.M.A.: Medical Politics Committee.

DEC. 20.—Queensland Branch, B.M.A.: Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide). All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

Editorial Notices.

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